

OTORHINOLARYNGOLOGY

Solved Question Papers

Third Edition

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Course Contents

■ DISEASES OF THE EAR

- Surgical anatomy
 - External, middle and inner ear
- Physiology of hearing and vestibular function
- Examination of the ear
 - Tuning fork tests, hearing assessment in children—broad outline, referred pain in the ear, otalgia, tinnitus
- Deafness
 - Types and causes
- Diseases of external ear
 - Perichondritis, otitis externa, cerumen, foreign body, furunculosis, keratosis
- Diseases of middle ear
 - Acute and chronic suppurative otitis media, obturans, otitis media with effusion, otosclerosis, cholesteatoma
- Audiometry
 - Pure tone
- Functional examination of inner ear (vestibule)
 - Calorie test, positional nystagmus test
- Deaf mutism
- Meniere's disease
- Complications of otitis media
 - Mastoiditis (acute and chronic), lateral sinus thrombosis, labyrinthitis, otogenic brain abscess
- Mastoidectomy
 - Principles
- Tumors of the ear
 - Glomus, acoustic neuroma.

■ DISEASES OF THE NOSE AND PARANASAL SINUSES

- Surgical anatomy and physiology of nose and paranasal sinuses, including olfaction
- Congenital diseases of the nose, cleft lip and palate and choanal atresia
- Diseases of external nose
 - Furunculosis, vestibulitis, rhinophyma, rodent ulcer
- Trauma to nose and paranasal sinus
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- Foreign body in nose
 - Classification of foreign bodies and management of animate and inanimate foreign bodies, rhinolith
- Causes of epistaxis and management
- Disease of nasal septum
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- Diseases of paranasal sinuses
 - Acute and chronic sinusitis, complications of sinusitis and management

- Sinonasal polyposis
 - Diagnosis and management
- Basic principles of functional endoscopic sinus surgery (FESS)
- Tumors of the nose and paranasal sinuses
 - Benign tumors like papilloma, inverted papilloma, fibrous dysplasia
 - Malignant tumors like squamous cell carcinoma, melanoma, olfactory neuroblastoma.

■ DISEASES OF THE NASOPHARYNX

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- Nasopharyngeal carcinoma
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 - Diagnosis and management
- Juvenile angiofibroma
 - Diagnosis and management.

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- Physiology of mastication
- Diseases of the tonsils
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 - Diagnosis and management
 - Vincent's angina
 - Diagnosis and management
 - Diphtheritic tonsillitis
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- Stridor
 - Causes of stridor and management
- Hoarseness of voice
 - History-taking, causes and management
- Chronic infections of larynx
 - Chronic nonspecific and specific laryngitis, granulomatous conditions of larynx
- Neurological infections of larynx
 - Cord palsy
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- Tumors of larynx
 - Diagnosis of laryngeal tumors and management.

■ DISEASES OF THE TRACHEA

- Surgical anatomy of trachea, stridor, tracheostomy in detail.

■ DISEASES OF THE ESOPHAGUS

- Surgical anatomy, physiology of deglutition
- Causes of dysphagia, diagnosis and management
- Diseases, such as congenital atresia, injuries (traumatic and chemical)
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- Neurological problems
- Oncology.

■ DISEASES OF THE BRONCHUS

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 - Indications, contraindications and complications of foreign body in bronchus.

UNIVERSITY EXAMINATION PATTERN

Eligibility for Writing the University Examination

The candidate should have at least 35% aggregate in the two of the three internals conducted by the college and should also have minimum 75% attendance in Theory and Practical classes conducted.

Criteria for Passing the University Examination

The candidate should secure minimum 50% in the university theory examination (University theory + Viva voce) and the university practical examinations separately. Internal assessment marks would not be considered for passing criteria, however, they would be added to final marks to determine class of passing.

Distribution of Marks

	Internal Assessment		University Examination	
	Maximum marks	Minimum marks to qualify	Maximum marks	Minimum marks to pass
Theory examination	30 marks	11 marks	100 marks	60 marks
Viva voce	—	—	20 marks	
Practical examination	20 marks	07 marks	80 marks	40 marks

Distribution of Marks for University Examination

Theory Examination

It is of 150 marks in total, which consist of:

University Examination	100 marks
University Viva Voce	20 marks
Theory Internals	30 marks

Clinical Examination

It is of 100 marks in total, which consist of:

University Clinical	80 marks
Clinical Internals	20 marks

Theory Examination

There shall be one theory paper carrying 100 marks. The pattern of questions would be of three types.

2 Long Essay Questions	2 × 10 marks	20 marks
10 Short Essay Questions	10 × 5 marks	50 marks
10 Short Answer Questions	10 × 3 marks	30 marks
Total		100 marks

Clinical Examination

Practical examination will carry 80 marks (2 cases of 40 marks each).

Viva Voce Examination

Viva voce carries 20 marks.

i. Instruments + viva	10 marks
ii. X-rays + viva	10 marks

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3. Nasal furuncle.	304	J15(RS3)
4. Vestibulitis of nose.	243	D12(RS3), D13(RS3), J12

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2. Causes and treatment of deviated nasal septum.	280	J14(RS3)
3. Nasal Septal hematoma.	350	J12, J14(OS)
4. Septal abscess.	312	D15(RS3)
5. Septal perforation (clinical features and management).	185	J11(RS2), D16(RS3), J07
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1. Anatomy of the nasal septum (bones taking part in the formation/labeled diagram).	341	D08, J15, D06(OS), J10(OS)
2. Chevallet fracture.	387	D16
3. Jarjavay fracture.	149	J10(RS2)
4. Hematoma septum (management).	350	D17(RS3), D00, D01, D04, J05, D05, D14, J09(OS)
5. Septal abscess (management).	312	D12(RS3), D02, J11, D13, J10(OS)
6. Nasal septal perforation (causes/treatment).	185	D07(RS2), D11(RS2), J17(RS3), J06, D12, J01(OS)
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1. Describe the etiology, pathology, clinical features, investigations and management of atrophic rhinitis.	289	D14(RS3), J03, J09, D10, J13, J15, J01(OS), J11(OS)
Short Essays		
1. Atrophic rhinitis (causes, clinical features and management/surgical treatment).	289	D13(RS3), J14(RS3), D15(RS3), D00, J01, D02, D04, D07, D12, J14, J17, J02(OS), D05(OS), D08(OS)
2. Nasal douching.	291	J14(OS)
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1. Mention causes of secondary atrophic rhinitis.	289	J16(OS)
2. Rhinitis caseosa.	242	D12(RS3), J07, D14
3. Rhinitis sicca.	189	J11(RS2), D16(RS3)
CHAPTER 28: GRANULOMATOUS DISEASES OF NOSE		
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1. What is rhinosporidiasis? Describe its pathology, clinical features and treatment.	141	D07
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1. Rhinoscleroma (etiology, stages, clinical features, investigations and management).	265	D13(RS3), J14(RS3), J07, D07, J09, D10, D12, D10(OS), J13(OS)
2. Fungal infections of nose and paranasal sinuses.	94	J05, D01(OS)
3. Nasal rhinosporidiosis (etiology, clinical features and management).	141	J10(RS2), D13(RS3), D01, J02, D06, D11, D02(OS), J07(OS), J09(OS)
4. Mucormycoses of nose and paranasal sinuses.	94	J09(RS2), D02
5. Midline granuloma.	94	J09(RS2)

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2. Rhinoscleroma—pathology.	265	D07(RS2), J16(RS3), J04, J08
3. Rhinosporidiosis—management.	141	J17(RS3), D03
CHAPTER 29: MISCELLANEOUS DISORDERS OF NASAL CAVITY		
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2. Rhinolith—etiology, clinical features and management.	70	J01, D01, J12, D06(OS)
3. Nasal myiasis.	22	D13
4. Choanal atresia.	271	D13(RS3)
5. Cerebrospinal fluid rhinorrhea (etiology, clinical features and management).	139	J10(RS2), D12(RS3), D14(RS3), J03, J16
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1. Rhinolith.	70	D08(RS2), J12(RS2), J13(RS3), J17(RS3), J07, J09, J15, J03(OS), D08(OS)
2. Nasal myiasis (management).	22	D07(RS2), J12(RS2), J14(RS3), J01, D07, J11, D09(OS)
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2. Allergic rhinitis (clinical features, management).	118	J02, D05, J11, J16
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2. Discuss about Little's area with diagram and its clinical importance.	190	D10, D15, D12(OS)
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1. Discuss the etiopathology, clinical features, diagnosis and management of juvenile nasopharyngeal angiofibroma (JNA).	102	D09(RS2), J11(RS2), D16(RS3), J01, D01, D08, D14, D12(OS)
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3. Describe the anatomy of retropharyngeal space. Discuss the etiopathogenesis, clinical features and management of (acute) retropharyngeal abscess.	105	D14(RS3), J07, J09(OS)
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2. Glottic cancers have high cure rate. Give reasons.	366	D00, J03, D04
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6. Investigations and treatment of glottic cancer.	161	J04
7. Treatment of carcinoma larynx (T1 lesion).	163	J00(OS)
8. Speech rehabilitation following laryngectomy.	378	D05

QUESTIONS	PAGE No.	EXAMINATIONS
Short Answers		
1. TNM classification of carcinoma larynx.	162	D07
2. T1 glottic malignancy.	162	D03
3. Esophageal voice.	378	D00, D02, D04, J05
CHAPTER 63: VOICE AND SPEECH DISORDERS		
Short Essays		
1. Discuss the management of an adult patient presenting with hoarseness of voice.	8	J17(RS3)
2. 'Hoarse voice'—causes (in 50 years old male).	7	D07(RS2), D13
3. Functional aphonia.	151	D05, D11
4. Puberphonia.	134	D07(OS)
5. Rhinolalia.	72	J10
Short Answers		
1. Enumerate causes of hoarseness in 50 years old female.	7	J10
2. Sudden aphonia.	151	D05(OS)
3. Puberphonia.	134	D09(RS2), J11, J15, D99(OS), J02(OS), D03(OS)
4. Rhinolalia.	72	D08(RS2)
5. Rhinolalia aperta (causes).	72	D01, J02, J13(OS)
6. Rhinolalia clausa.	72	J03, J16, J05(OS)
CHAPTER 64: TRACHEOSTOMY AND OTHER PROCEDURES FOR AIRWAY MANAGEMENT		
Long Essay		
1. Tracheostomy—define, indications, surgical procedure, postoperative care and complications.	9	J05(OS), D06(OS), J07(OS), J16(OS)
Short Essays		
1. Advantages of tracheostomy over endotracheal intubation.	12	D06
2. Tracheostomy—types, indications (nonobstructive), procedure and complications.	9	D07(RS2), D08(RS2), J11(RS2), D12(RS3), J15(RS3), D16(RS3), J17(RS3), J04, D04, J08, J10, D14, J17, D00(OS), D07(OS), D12(OS)
3. Postoperative management after tracheostomy.	11	J03(OS)
4. What is emergency tracheostomy? Name the indications for it.	9	D02
Short Answers		
1. Indications for tracheostomy.	9	D10(RS2), J12
2. Nonobstructive indication for tracheostomy.	9	D15
3. Decannulation of tracheostomy.	12	D11
4. Causes of decanulation problem.	12	D03
5. Complications of tracheostomy.	12	D05, J08, J16
6. Cricothyrotomy.	369	D01(OS)
CHAPTER 65: FOREIGN BODIES IN AIR PASSAGE		
Short Essay		
1. Foreign body in bronchus.	184	J11(RS2), D16(RS3), J13, D16

QUESTIONS	PAGE No.	EXAMINATIONS
SECTION VI: THYROID GLAND AND ITS DISORDERS		
CHAPTER 66: THYROID GLAND AND ITS DISORDERS		
Short Essay		
1. Lingual thyroid.	34	J08(RS2)
Short Answer		
1. Lingual thyroid.	34	J12(RS2), J13(RS3)
SECTION VII: DISEASES OF ESOPHAGUS		
CHAPTER 67: ANATOMY AND PHYSIOLOGY OF ESOPHAGUS		
Long Essay		
1. Discuss the physiological basis and mechanism (phases) of deglutition.	44	D08(RS2), J14
Short Essay		
1. Describe the mechanism of deglutination.	44	J01(OS)
Short Answer		
1. Constrictions of esophagus.	314	D15(RS3)
CHAPTER 68: DISORDERS OF ESOPHAGUS		
Short Essays		
1. Esophageal perforation.	21	J16(OS)
2. Plummer-Vinson (Paterson Brown Kelly) syndrome.	292	D14(RS3), D05, J07, D13, J07(OS), D09(OS), J10(OS)
3. Globus hystericus.	233	D06
4. Achalasia cardia.	379	J07, D00(OS), D07(OS), J12(OS)
Short Answers		
1. Esophageal perforations.	21	D07(RS2)
2. Plummer-Vinson (Patterson Kelly) syndrome.	292	J15(RS3), J08, J03(OS), J16(OS)
3. Globus pharynges (globus hystericus).	233	J12(RS2), J13(RS3), J11
4. Achalasia cardia.	379	D03(OS)
CHAPTER 69: DYSPHAGIA		
Long Essay		
1. How will you manage a case of 50 years old woman presenting with dysphagia.	173	D15
Short Essays		
1. Dysphagia—causes/management.	173	D10(RS2), D17(RS3), J02, J15, J02(OS), D02(OS)
2. Dysphagia lusoria.	174	D11
Short Answer		
1. Dysphagia lusoria.	174	J11(OS)
CHAPTER 70: FOREIGN BODIES IN FOOD PASSAGE		
Short Essay		
1. Clinical features and management of foreign bodies in esophagus.	380	J09
Short Answers		
1. Foreign body in cricopharynx.	380	D10

QUESTIONS	PAGE No.	EXAMINATIONS
2. Foreign body esophagus.	380	D01, J12(OS)
3. Management of ingested foreign body.	381	J06
SECTION VIII: RECENT ADVANCES		
CHAPTER 71: LASER SURGERY, RADIOFREQUENCY SURGERY, HYPERBARIC OXYGEN THERAPY AND COBLATION		
Short Answer		
1. Applications of lasers in ENT.	178	D10(RS2), J14(RS3), J09
CHAPTER 72: CRYOSURGERY		
Short Essay		
1. Cryosurgery in otorhinolaryngology.	390	J12
CHAPTER 73: RADIOTHERAPY IN HEAD AND NECK CANCER		
Short Answer		
1. Enumerate complications of radiotherapy.	371	J09
CHAPTER 74: CHEMOTHERAPY IN HEAD AND NECK CANCER		
Short Answers		
1. Chemotherapy.	73	D08(RS2)
2. Palliative treatment.	73	D08(RS2)
CHAPTER 75: HIV INFECTIONS/AIDS AND ENT MANIFESTATIONS		
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1. Manifestation of AIDS in ENT.	8	D07(RS2), D13
Short Answer		
1. ENT manifestations in acquired immunodeficiency syndrome.	8	D10(RS2)
SECTION IX: CLINICAL METHODS IN ENT AND NECK MASSES		
CHAPTER 76: CLINICAL METHODS IN ENT		
Short Essays		
1. Enumerate cause of trismus. Discuss management of one common cause.	319	D07
2. Indirect laryngoscopy.	316	J06(OS)
Short Answers		
1. Draw a neat labeled diagram of anterior rhinoscopy.	149	D12(OS)
2. Anterior rhinoscopy.	149	J10(RS2)
3. Posterior rhinoscopy.	388	D15
4. Diagram of structures seen on posterior rhinoscopy.	388	J14
5. Enumerate causes of trismus.	319	D15(RS3), D08, J13(OS)
6. Indirect laryngoscopy.	316	D15(RS3), D00, D02, D04, J16, J05(OS)
7. Diagrammatic view of structures seen on Indirect laryngoscopy.	317	D13
CHAPTER 77: NECK MASSES		
Short Essays		
1. Give differential diagnosis of lymph node in neck.	391	J08
2. Differential diagnosis of a midline neck swelling.	240	D12(RS3), D13(RS3), D14(RS3)
3. Thyroglossal cyst.	223	J12(RS2), J13(RS3), D01, D02, D13

QUESTIONS	PAGE No.	EXAMINATIONS
4. Differential diagnosis of lateral neck swellings.	183	J11(RS2), D16(RS3)
5. Branchial cyst.	188	J13
Short Answers		
1. Thyroglossal cyst.	223	D15(RS3), J09
2. Branchial cyst.	188	J11(RS2), D16(RS3)
3. Plunging ranula.	187	D14(RS3)
SECTION X: OPERATIVE SURGERY		
CHAPTER 78: MYRINGOTOMY		
Short Essay		
1. Myringotomy—indications, procedure and complications.	237	J02, D07
Short Answers		
1. Indications for gromet insertion in ear.	237	J05
2. Myringotomy.	237	D07(RS2), J06(OS)
3. Indications and sites for myringotomy.	237	J14
CHAPTER 79: EAR SURGERY AND APPROACHES		
Short Answer		
1. Describe the types of mastoidectomies.	371	J03
CHAPTER 80: CORTICAL MASTOIDECTOMY		
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1. Define coalescent mastoiditis. Describe cortical mastoidectomy.	230	J12(RS2), J13(RS3), D12, J13(OS)
Short Answers		
1. Simple mastoidectomy.	371	D15
2. Complications of mastoidectomy.	99	J09(RS2), J13
CHAPTER 81: RADICAL MASTOIDECTOMY		
None		
CHAPTER 82: MODIFIED RADICAL MASTOIDECTOMY		
Short Essay		
1. What is modified radical mastoidectomy? Mention its indications and complications.	371	J16(OS)
Short Answer		
1. Modified radical mastoidectomy.	371	D16
CHAPTER 83: MYRINGOPLASTY		
Short Essay		
1. Myringoplasty.	196	D10
Short Answers		
1. Myringoplasty.	196	J09
2. Graft materials for myringoplasty.	195	J17
CHAPTER 84: PROOF PUNCTURE (ANTRAL IRRIGATION)		
Short Essay		
1. Antral lavage/antral wash.	12	D07(RS2), J12, D15

QUESTIONS	PAGE No.	EXAMINATIONS
Short Answer		
1. Antral puncture (indications and complications).	12	D07, D01(OS)
CHAPTER 85: INTRANASAL INFERIOR MEATAL ANTROSTOMY		
None		
CHAPTER 86: CALDWELL-LUC OPERATION		
Short Essay		
1. Caldwell-Luc's operation—indications, procedure (steps) and complications.	344	J03, J08, J03(OS), J05(OS)
Short Answer		
1. Indications for Caldwell-Luc operation.	344	J17(RS3), D08, D13, J09(OS)
CHAPTER 87: SUBMUCOUS RESECTION OF NASAL SEPTUM (SMR OPERATION)		
Short Essays		
1. Discuss the difference between septoplasty and submucous resection of nasal septum (SMR) operation.	143	J10(RS2), D00, J01, D04, D05, J13, D14
2. Indications and complications of SMR.	282	D03, J16
3. Discuss complications of submucous resection of nasal septum.	284	J08
4. Discuss complications and their management of nasal septal surgery. Describe septal abscess.	284, 286, 312	D10, J00(OS)
Short Answers		
1. Indications for submucous resection of nasal septum.	282	J10
2. Complications of submucosal resection.	284	J08(RS2)
CHAPTER 88: SEPTOPLASTY		
Short Answer		
1. Septoplasty	284	D16
CHAPTER 89: DIAGNOSTIC NASAL ENDOSCOPY		
Short Answer		
1. Diagnostic nasal endoscopy (technique).	100	J09(RS2), D10(RS2), J14
CHAPTER 90: ENDOSCOPIC SINUS SURGERY		
Short Essay		
1. Endoscopic sinus surgery.	15	J16(RS3)
CHAPTER 91: DIRECT LARYNGOSCOPY		
Short Essays		
1. Mention the indications for direct laryngoscopy.	381	J11(OS)
2. Indications and contraindications of rigid laryngoscopy.	381	J01, J03
Short Answer		
1. Indications for direct laryngoscopy.	381	J05
CHAPTER 92: BRONCHOSCOPY		
Short Essays		
1. Bronchoscopy (indications, contraindications and complications).	200	D11(RS2), J17(RS3), D12, J07(OS), D09(OS), J10(OS)
2. Indications and contraindications of rigid bronchoscopy.	200	D03, J01(OS)

QUESTIONS	PAGE No.	EXAMINATIONS
CHAPTER 93: ESOPHAGOSCOPY		
Short Essays		
1. Esophagoscopy (indications and complications).	222	J12(RS2), J13(RS3), D13(RS3), J11, D16
2. Indications and contraindications of rigid esophagoscopy.	222	J04, J15, D08(OS)
Short Answers		
1. Esophagoscopy.	222	J11, D00(OS)
2. Two indications for esophagoscopy.	222	D11(OS)
3. Complications of rigid esophagoscopy.	223	J06
CHAPTER 94: TONSILLECTOMY		
Short Essays		
1. Tonsillectomy—indications and complications.	171	D10(RS2), D13(RS3), J07, J13, J14, D15
2. Briefly describe the operative steps of dissection method of tonsillectomy.	172	J06
3. Primary hemorrhage during tonsillectomy.	173	D06(OS)
4. Secondary hemorrhage.	302	J15(RS3)
Short Answers		
1. Enumerate the complications of tonsillitis.	173	J14
2. Immediate complications of tonsillectomy.	173	J17
3. Post-tonsillectomy bleeding.	173	J03(OS), J05(OS)
4. Reactionary hemorrhage after tonsillectomy.	173	D00, J01, D04, J00(OS), J01(OS), D11(OS)
5. Management of reactionary hemorrhage following tonsillectomy.	173	J14(RS3)
6. Secondary hemorrhage following tonsillectomy—causes and treatment.	173	D07, D06(OS), D09(OS), D10(OS)
CHAPTER 95: ADENOIDECTOMY		
Short Essay		
1. Adenoidectomy (indications and complications).	324	D14(RS3), J09, J06(OS)
Short Answers		
1. Complications of adenoidectomy.	325	D06
2. Post-adenoidectomy hemorrhage.	325	J02
CHAPTER 96: RADIOLOGY IN ENT		
None		
MISCELLANEOUS		
Short Essays		
1. Tracheostomy tubes.	326	J16(RS3), D01, J05, J13
2. Describe features of ideal tracheostomy tube.	328	D07
3. Glandular fever.	300	J15(RS3)
4. Write a note on MLS (micro-laryngeal surgery).	364	D00, J01, D04
5. What are the causes of unilateral nasal discharge?	382	J02, J08
6. Mention the branches of external carotid artery.	96	J05
7. Indications and technique of microlaryngoscopy.	364	J06
8. TNM staging and its clinical application.	32	J08(RS2)
9. Bronchopulmonary segments.	37	J08(RS2)

QUESTIONS	PAGE No.	EXAMINATIONS
10. Anaerobic infection in ENT.	91	J09(RS2)
11. Occult primary.	392	J09
12. Role of local anesthesia in otorhinolaryngology.	56	D08(RS2)
13. Nasal decongestants.	56	D08(RS2)
14. Newer generation antihistaminics.	92	J09(RS2)
15. Dangerous area of the face.	226	J12(RS2), J13(RS3)
Short Answers		
1. Tracheostomy tubes.	326	D11(RS2), D14(RS3)
2. External carotid artery branches.	96	J09(RS2), D10
3. Carcinoma-in-situ.	134	D09(RS2)
4. Post-nasal drip.	149	J10(RS2)
5. Mucociliary dyskinesia.	177	D10(RS2)
6. Cardiospasm.	379	D10
7. Compare indirect and direct laryngoscopy.	370	D10
8. Nasal decongestants.	56	D11(RS2)
9. Cold abscess of neck.	244	D12(RS3)

MBBS PHASE III EXAMINATION

DECEMBER 2007

(Revised Scheme 2)

■ LONG ESSAYS

1. Describe the physiology and mechanism of hearing.

- Hearing is one of 5 special senses and main function of ear.

Mechanism of Hearing

- Mechanism of hearing can be described as follows:
 - Mechanical conduction of sound (conductive apparatus)
 - Transduction of mechanical energy into electrical impulses (sensory perceiving apparatus)
 - Conduction of electrical impulse to brain (neural apparatus).

Conduction of Sound

A. External ear

- External auditory canal directs sound entering it towards tympanic membrane
- Pinna in humans has lost most of its sound collection function.

B. Middle ear

- Sound wave falling on tympanic membrane set it into vibration which are transmitted to through ossicular chain to oval window by foot plate of stapes
- Middle ear contents have certain important functions in conduction of sound

a. Impedance matching (magnification of sound)

- These are mechanisms by which middle ear amplifies incoming sound to compensate for loss of 30 dB sound occurring at air and fluid interphase.

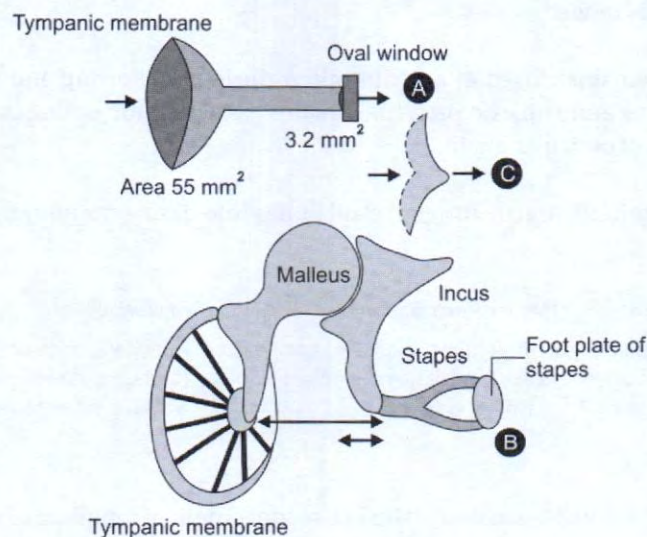


Figure 1: Impedance matching

<i>Hydraulic action of tympanic membrane</i>	<i>Lever action of ossicles</i>	<i>Buckling factor</i>
<ul style="list-style-type: none"> Effective vibratory area of tympanic membrane is about 14 times greater than area of oval window thus all force collected over tympanic membrane is concentrated on small oval window Thus pressure is increased by: $\frac{\text{Area of tympanic membrane}}{\text{Area of oval window}} = \frac{55 \text{ mm}^2}{3.2 \text{ mm}^2} = 17 \text{ times}$ This is most important factor in achieving impedance matching 	<ul style="list-style-type: none"> Arm of incus is shorter than handle of malleus and this produces a lever action that increases force and decreases velocity at stapes This increases pressure by 1.30 times 	<ul style="list-style-type: none"> This depends on the conical shape of tympanic membrane As membrane moves in and out, it buckles so that handle of malleus moves less than surface of membrane This again increases force and decreases velocity This also increases pressure to some extent

b. Phase difference between oval and round window

- There exists a phase difference of 4 dB between oval window and round window, i.e. sound waves striking tympanic membrane do not reach both oval and round window simultaneously
- Sound reaches first to oval window due to preferential ossicular chain thus when oval window is perceiving wave of compression, round window is at phase of rarefaction
- This is very essential for conduction of sound because if sound wave strikes both window simultaneously, there would be cancelation of their effect with no movement of perilymph and thus hearing
- This is achieved by intact tympanic membrane and cushion of air in middle ear around round window.

c. Natural resonance of external and middle ear

- Inherent anatomic and physiological properties of external and middle ear allow certain frequencies of sound to pass more easily to inner due to their natural resonance

	<i>Natural resonance</i>
♦ External auditory canal	3000 Hz
♦ Tympanic membrane	800–1600 Hz
♦ Middle ear	800 Hz
♦ Ossicular chain	500–2000 Hz

- This greatest sensitivity of sound transmission is between 500–3000 Hz and most of day to day conversations of humans fall into this range.

C. Eustachian tube

- Auditory tube usually remains closed at rest but opens during swallowing and yawning when it equalizes air pressure in middle ear to atmospheric pressure which is essential for optimum functioning of ear drum and conducting mechanism of ossicular chain.

D. Bone conduction

- Sound sounds are transmitted directly through skull bones into basilar membrane and cochlear fluid.

Mechanism

<i>Translatory or inertial mechanism</i>	<i>Compression mechanism</i>	<i>Effect of mandible</i>
♦ During vibration of skull, ossicles lag behind due to inertia causing movement of stapes relative to oval window	♦ Vibration of skull sets cochlear fluid and basilar membrane into vibration	♦ During vibration of skull, mandible lags behind and sets cartilaginous external auditory canal into vibration which is then transmitted to inner ear

Perception of sound

- Sound wave transmitted through ossicular chain cause movement of footplate of stapes which sets perilymph in cochlea in vibration
- This moves basilar membrane, setting up sharing force between tectorial membrane and hair cells

- Distortion of hair cells gives rise to cochlear microphonics which trigger nerve impulse
- Sound wave, depending upon its frequency reaches maximum amplitude on particular place on basilar membrane and stimulates that segment
- Representation of frequencies is such that higher frequencies are represented in basal turn of cochlea and then progressively lower ones towards apex.

Neural pathway

- Hair cells present in organ of Corti are innervated by bipolar cells of spiral ganglion which collectively form cochlear nerve and relay information from inner ear to ventral and dorsal cochlear nuclei
- From here, both crossed and uncrossed fibers travel to superior olivary nucleus, lateral lemniscus, inferior colliculus, medial geniculate body finally reaching auditory cortex of temporal lobe.

Physiology of Hearing (Theories of Hearing)

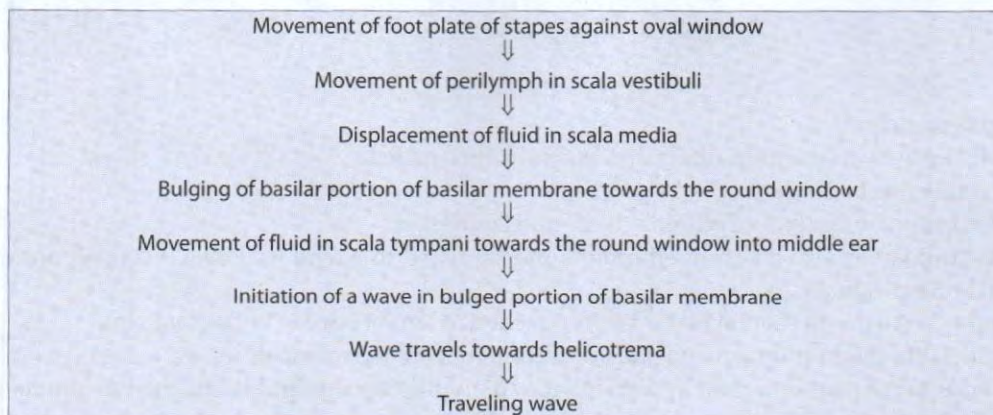
- Many theories have been postulated to explain physiology of hearing

Telephone theory (Rutherford)	Place theory (Helmoltz)	Volley theory (Wever)	Travelling wave theory (Bekesy)
Whole basilar membrane vibrates with each sound and perception of pitch is related to rate of firing of individual nerve fibers	Perception of pitch depends upon selective vibration of a specific place on basilar membrane	Combines both telephone and place theory to suggest that higher frequencies are perceived by place mechanism whereas lower frequencies are perceived by telephone mechanism	Sound waves starting from oval window produce a wave that travels along basilar membrane to apex of cochlea, increasing in amplitude as moves and finally dies away

- Of various theories put forward, Von Bekesy's travelling wave theory best explains mechanism of hearing by human ear.

Travelling Wave Theory

- This theory was proposed by Von Bekesy after studying physical properties of basilar membrane like stiffness, dimension and movement in response to a sound frequency under a microscope using stroboscopic illumination
- He showed that membrane is narrower and stiffer at base and wider and less stiff at apex
- He demonstrated displacement of membrane at basal regions with a high frequency and entire membrane with a lower frequency
- Movement of foot plate of stapes against oval window causes movement of perilymph in scala vestibule which causes displacement of fluid in scala media as vestibular membrane is flexible
- This causes bulging of basal portion of basilar membrane towards round window, which in turn, moves fluid in scala tympani towards round window into middle ear
- Elastic tension developed in basilar fibers in bulged portion of basilar membrane initiates a wave which travels along basilar membrane towards helicotrema
- This is called travelling wave



- Though this wave is weak when originated, it becomes stronger as it travels from base to apex

- At point where wave is strongest on basilar membrane is called resonance point and wave does not travel any further from here
- Distance between stapes and resonance point is inversely proportional to frequency of sound waves reaching ear
- High pitched sound generates waves, which dies near base of cochlea whereas medium frequency sound waves reach halfway and low frequency sound waves travel entire distance of basilar membrane
- Movement of basilar membrane also causes movement of hairs leading to excitement of hair cells thus generating action potential.

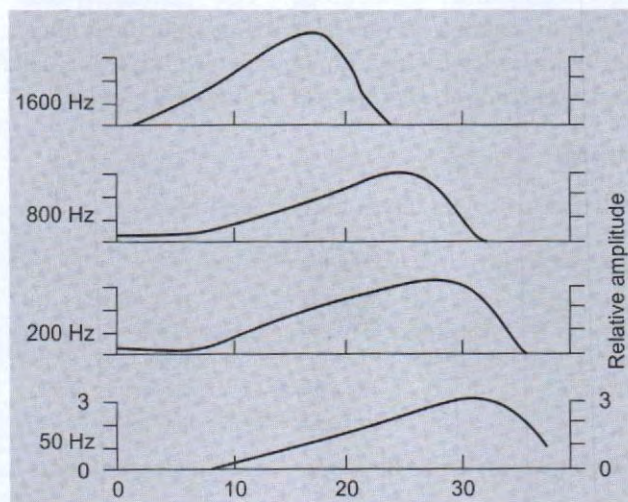


Figure 2: Travelling waves

2. How do you manage a stage III carcinoma larynx?

Refer Question No. 2 December 2010 (RS2).

SHORT ESSAYS

3. Describe lateral wall of nasal cavity.

- Lateral wall of nose is very important because all paranasal sinuses open into it.

Formation (Formed by following bones)

Anteriorly	In middle	Posteriorly
<ul style="list-style-type: none"> ♦ Ascending process of maxilla ♦ Nasal bone 	<ul style="list-style-type: none"> ♦ Ethmoid in upper part ♦ Medial wall of maxilla in middle ♦ Inferior turbinate in lower part 	<ul style="list-style-type: none"> ♦ Perpendicular plate of palatine bone ♦ Medial pterygoid plate

Features

- Nasal turbinates (conchae)
 - Three shelf like bony projections directed downward and medially
 - Covered by mucous membrane
 - Superior turbinate is a part of ethmoid bone and is smallest.
 - Middle turbinate is also a part of ethmoid bone, attached to lateral wall by a S-shaped bony lamella called ground or basal lamella
 - * Anterior 1/3rd lies in sagittal plane and is attached to lateral edge of cribriform plate
 - * Middle 1/3rd lies in frontal plane and is attached to lamina papyracea
 - * Posterior 1/3rd runs horizontally forming roof of middle meatus and is attached to lamina papyracea and medial wall of maxillary sinus.
 - Inferior turbinate is a separate bone.

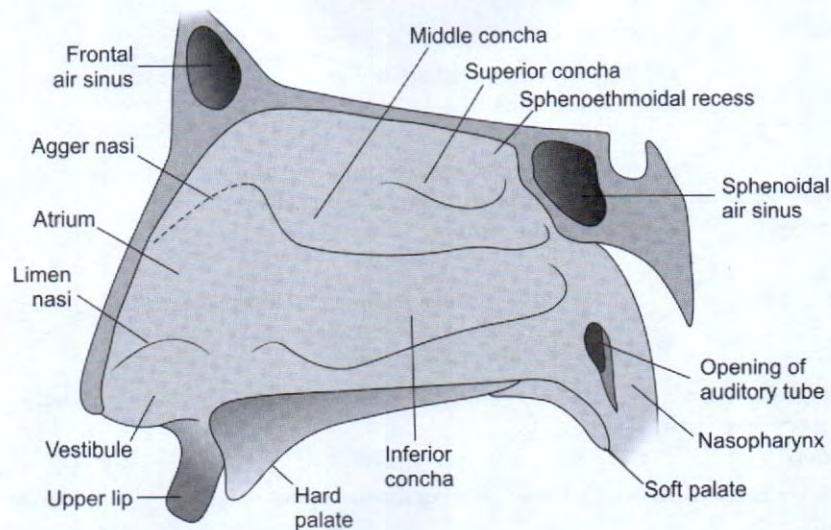


Figure 3: Lateral wall of nasal cavity—formation

b. Meatuses

- Three passages under three respective turbinates
 - i. Superior meatus is smallest
 - ii. Middle meatus or osteomeatal complex presents following features:
 - * Bulla ethmoidalis is a rounded projection
 - * Hiatus semilunaris is a semicircular sulcus running anterior and inferior to bulla
 - * Infundibulum is a small funnel-shaped passage anterior to bulla.
 - iii. Inferior meatus is largest meatus.

c. Other features

- Sphenoethmoidal recess is a triangular fossa above superior meatus
- Atrium is a shallow depression in front of middle meatus lying above vestibule and below aggar nasi
- Vestibule is portion lined by skin, medial to ala of nose.

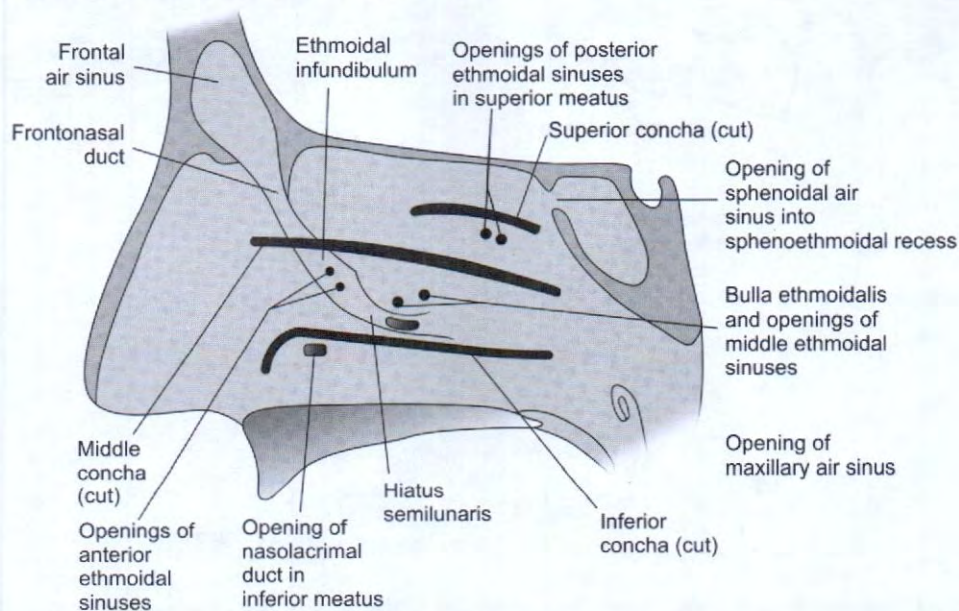
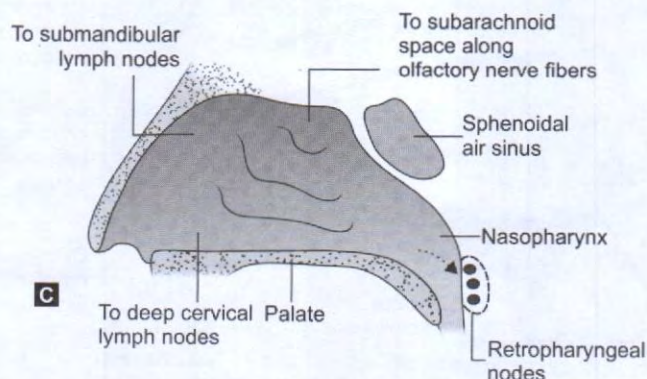
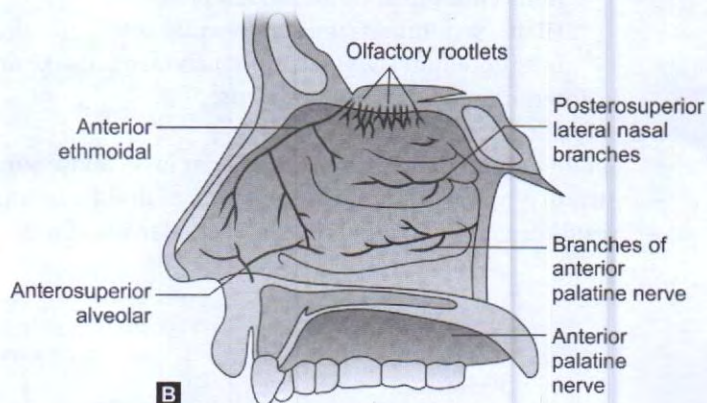
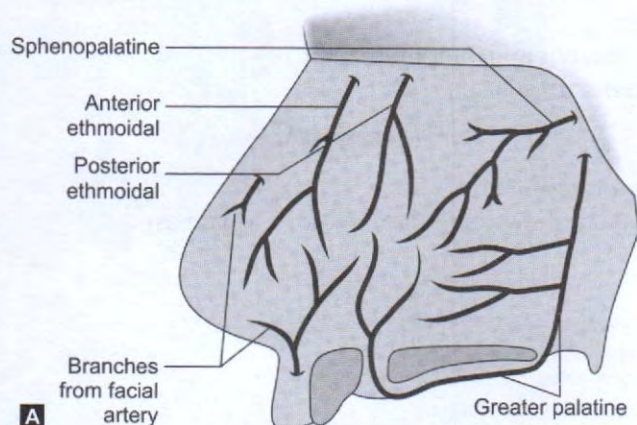


Figure 4: Lateral wall of nasal cavity—openings

Openings

- | | |
|---------------------------|---|
| i. Superior meatus | • Posterior ethmoidal sinuses |
| ii. Middle meatus | • Frontal sinus |
| | • Maxillary sinus |
| iii. Inferior meatus | • Anterior and middle ethmoidal sinuses |
| iv. Sphenothmoidal recess | • Nasolacrimal duct guarded by Hasner's valve |
| | • Sphenoidal sinus |

Quadrants	Arterial supply	General nerve supply: Trigeminal nerve Special sensory: Upper part of lateral wall by olfactory nerve	Lymphatic drainage
Anterosuperior	Anterior ethmoidal artery Posterior ethmoidal artery Facial artery	Anterior ethmoidal nerve	Submandibular nodes
Anteroinferior	Branches from facial and greater palatine arteries	Anterior superior alveolar nerve	Deep cervical nodes
Posterosuperior	Sphenopalatine artery	Posterior superior lateral nasal branches from pterygopalatine ganglion	Subarachnoid space along with olfactory nerve fibers
Posteroinferior	Branches of greater palatine artery	Anterior palatine branch from pterygopalatine ganglion	Retropharyngeal nodes



Figures 5A to C: (A) Lateral wall of nasal cavity—arterial supply; (B) Lateral wall of nasal cavity—nerve supply; (C) Lateral wall of nasal cavity—lymphatic drainage

Significance

- Infection of nose may spread through cribriform plate to anterior cranial fossa and to lacrimal apparatus through nasolacrimal duct
- Hypertrophy of mucosa over inferior nasal concha is a feature of allergic rhinitis.

4. Cholesteatoma.

Refer Question No. 1 June 2013 (RS2).

5. What are the causes of 'hoarse voice'?

- Hoarse voice (hoarseness) is rough and unpleasant voice resulting from variations of periodicity and/or intensity of consecutive sound waves.

Etiology

a. Inflammations

<i>Acute</i>	<i>Chronic</i>
<ul style="list-style-type: none"> ♦ Acute laryngitis (following cold, influenza, exanthematous fever) ♦ Laryngotracheobronchitis ♦ Diphtheria 	<ul style="list-style-type: none"> ♦ Tuberculosis ♦ Syphilis ♦ Scleroma ♦ Fungal infections ♦ Chronic laryngitis ♦ Atrophic laryngitis

b. Tumors

<i>Benign</i>	<i>Malignant</i>	<i>Tumor like mass</i>
<ul style="list-style-type: none"> ♦ Papilloma (solitary and multiple) ♦ Hemangioma ♦ Chondroma ♦ Fibroma ♦ Leukoplakia 	<ul style="list-style-type: none"> ♦ Carcinoma 	<ul style="list-style-type: none"> ♦ Vocal nodule ♦ Vocal polyp ♦ Angiofibroma ♦ Amyloid tumor ♦ Contact ulcer ♦ Cysts ♦ Laryngocele

c. Trauma

- Misuse or overuse of voice
- External injuries to larynx (strangulations, cut throat, etc.)
- Submucosal hemorrhage
- Inhalation of irritant fumes
- Foreign bodies
- Intubation.

d. Paralysis

- Recurrent laryngeal nerve paralysis
- Superior laryngeal nerve paralysis.

e. Fixation of cords

- Arthritis or fixation of cricoarytenoid joints.

f. Congenital

- Laryngeal web
- Laryngeal cyst
- Laryngocele.

- g. Miscellaneous
- Dysphonia plica ventricularis
 - Myxedema
 - Gout.
- h. Functional
- Hysterical aphonia.

Investigations

Indirect laryngoscopy	Direct laryngoscopy or flexible fiberoptic laryngoscopy	Microlaryngoscopy	Bronchoscopy and esophagoscopy	Radiology
♦ Reveals many local laryngeal causes	♦ Confirms any local laryngeal causes and helps to obtain laryngeal swab for smear and culture	♦ Helps in precise diagnosis and obtain biopsy of lesion or assessment mobility of cricoarytenoid joints	♦ To exclude malignancy in paralytic lesions of cord	♦ X-ray (plain and contrast) and CT scans to confirm diagnosis

Treatment

Supportive	Conservative	Operative	Specific
♦ Voice rest ♦ Steam inhalation (of tincture benzoin, menthol, camphor, etc.) to reduce congestion ♦ Speech therapy (to teach patient to speak in relaxed manner without straining vocal cords)	a. Antibiotics – In case of inflammatory conditions b. Anti-inflammatory drugs – To reduce congestion and edema	♦ Phonosurgery	♦ Treatment of underlying cause

6. Middle ear contents and their functions.

Refer Question No. 1 June 2012 (RS2).

7. Blow out fracture of orbit.

Refer Question No. 3 December 2010 (RS2).

8. Manifestation of AIDS in ENT.

- AIDS or acquired immunodeficiency syndrome is caused by HIV (human immunodeficiency virus).

Pathogenesis

- It reduces the body's immune mechanism resulting in opportunistic infections by viruses, bacteria, fungi, protozoa and also activates neoplastic process resulting in numerous clinical conditions including in head and neck region.

Manifestations of AIDS in ENT

Ear	Nose	Oral cavity	Parotids	Esophagus	Neck
♦ Otitis media ♦ Kaposi's sarcoma of pinna ♦ Sensorineural hearing loss (due to cytomegalovirus affection of inner ear or 8th cranial nerve) ♦ Facial paralysis	♦ Chronic rhinitis ♦ Sinusitis (by aerobic and anaerobic bacteria) ♦ Fungal sinusitis (by aspergillus or mucormycosis)	♦ Thrush (white cheesy exudates on an erythematous mucosa in posterior oropharynx due to candida infection) ♦ Angular cheilitis ♦ Recurrent aphthous ulcer ♦ Hairy leukoplakia (white patches on lateral border of tongue due to EB virus infection) ♦ Kaposi's sarcoma (especially of palate) ♦ Non-Hodgkin's lymphoma	♦ Parotid cysts ♦ Parotitis	♦ Esophagitis (due to candida, CMV or HSV infection) ♦ Kaposi's sarcoma ♦ Lymphoma	♦ Cervical lymphadenopathy

9. Tracheostomy.

- Tracheostomy is a surgical opening in anterior tracheal wall which is converted into stoma on skin surface by inserting a tube.

Types

Emergency	Elective or routine (tranquil)		Permanent
Done urgently to establish airway in presence of complete or almost complete airway obstruction	Temporary tracheostomy done after thorough planning with availability of all necessary surgical facilities and instruments		Done in cases of bilateral abductor paralysis or laryngeal stenosis
	<i>Therapeutic</i>	<i>Prophylactic</i>	
	To relieve respiratory obstruction, remove tracheobronchial secretions or give assisted ventilation	To guard against anticipated respiratory obstruction or aspiration of blood or pharyngeal secretions	

Location

	High tracheostomy	Mid tracheostomy	Low tracheostomy
Location	At 1st tracheal ring, above level of thyroid isthmus	At level of 2nd or 3rd tracheal ring	Below level of thyroid isthmus
Indications	Carcinoma of larynx when laryngectomy is anticipated	Most preferred site for routine tracheostomy	Laryngeal papillomatosis to avoid implantation Enlarged thyroid isthmus
Disadvantages	Damage to 1st tracheal ring and causes perichondritis of cricoid cartilage and subglottic stenosis		Chances of injury to several large vessels lying close to trachea and impingement of tracheostomy tube on suprasternal notch

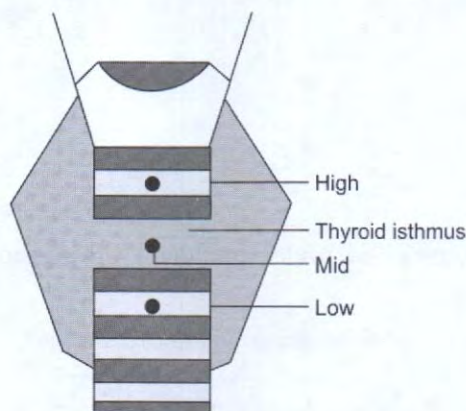


Figure 6: Tracheostomy—location (types)

Indications

Obstructive			Non-obstructive (All 'A's)
Respiratory obstruction	Retained secretion	Respiratory insufficiency	
a. Infections <ul style="list-style-type: none"> Acute laryngotracheobronchitis^a Acute epiglottitis^a Diphtheria^a Ludwig's angina Peritonsillar abscess Retropharyngeal abscess Parapharyngeal abscess Tongue abscess 	a. Inability to cough <ul style="list-style-type: none"> Coma of any cause (head injuries, cerebrovascular accidents, narcotic overdose) Paralysis of respiratory muscles (spinal injuries, polio, GB syndrome, myasthenia gravis) Spasm of respiratory muscles (tetanus, eclampsia, strychnine poisoning) 	<ul style="list-style-type: none"> Chronic lung conditions (emphysema, chronic bronchitis, bronchiectasis, atelectasis) Respiratory paralysis Severe chest injuries, flail chest 	<ul style="list-style-type: none"> Assisted ventilation Aspiration Anesthesia Alaryngeal Atelectasis

Contd...

Contd...

b. Trauma

- External injury to larynx and trachea*
- Cut throat injury to larynx*
- Strangulation and crush injury to larynx*
- Trauma due to endoscopies*
- Fracture of mandible
- Maxillofacial injuries

c. Neoplasms

- Benign and malignant neoplasms of URT, tongue and thyroid

d. Foreign body

- Laryngeal foreign body

e. Laryngeal edema*

- Due to steam, irritant fumes, allergy, radiation

f. Neurological

- Bilateral abductor paralysis*

g. Congenital anomalies

- Laryngeal web and cysts*
- Tracheoesophageal fistula*
- Bilateral choanal atresia*
- Glottis web*
- Subglottic stenosis or hemangioma*

b. Painful cough

- Chest injuries
- Multiple rib fractures
- Pneumonia

c. Aspiration of pharyngeal secretions

- Bulbar polio
- Polyneuritis
- Bilateral laryngeal paralysis

Note: Indications in infants*—Underlined; Indications in children*—Italicized

Contraindications

- Bleeding disorders
- General debility
- Diabetes and hypertension.

Procedure*Position*

- Supine with extended neck (by placing a pillow under shoulders) with surgeon standing on right of patient.

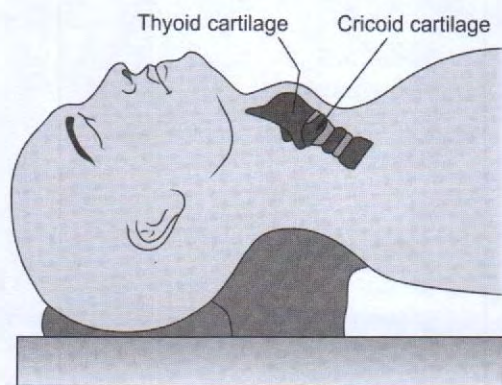


Figure 7: Tracheostomy—position

Anesthesia

- Not required (in emergency and unconscious patients)
- Local anesthesia with 1–2% lignocaine with epinephrine (elective tracheostomy).

Incision

- A vertical incision in midline of neck, extending from cricoids cartilage to just above sternal notch—in emergency and for beginners
- 5 cm long transverse incision, 2 fingers breadth above sternal notch (in elective tracheostomy for cosmetically better scar)—for elective surgeries

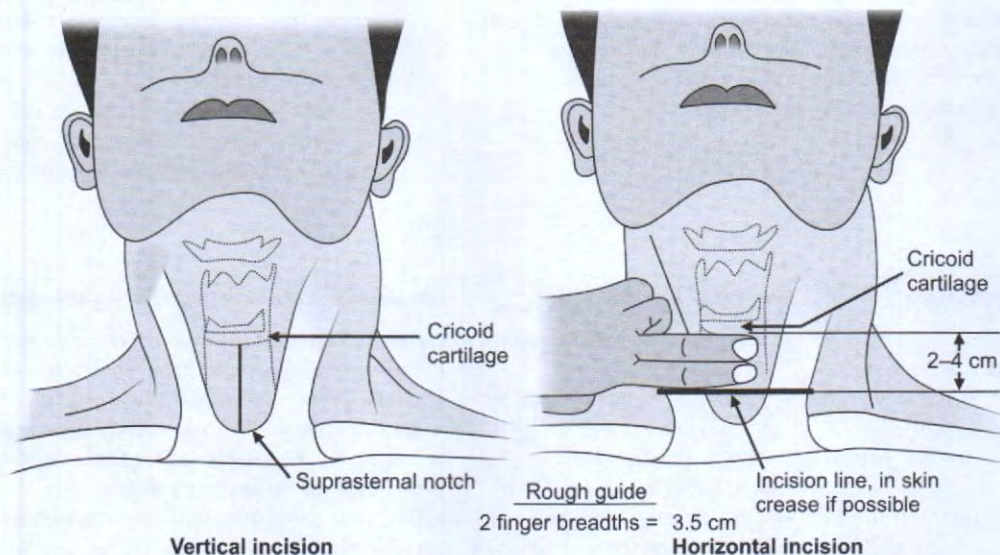


Figure 8: Tracheostomy—incisions

Steps

- Make desired incision
- Dissect tissue in midline
- Separate and retract strap muscles laterally using C-shaped or right angled retractors
- Incise pretracheal fascia below isthmus and expose trachea
- Thyroid isthmus is displaced upward (for low tracheostomy) or divided between clamps and suture ligated (for mid tracheostomy)
- Achieve complete hemostasis to prevent tricking of blood into trachea
- Inject few drops of 4% lignocaine into trachea (to suppress cough while incising trachea)
- Fix trachea with hook and opened with a vertical incision in region of 3rd-4th or 2nd or 3rd ring
- Convert vertical tracheal opening into a circular opening
- Insert tracheostomy tube of appropriate size and secure it by tapes
- Do not suture skin incision or pack it tightly (to prevent development of subcutaneous emphysema)
- Place gauze dressing between skin and flange of tube around stoma.

Postoperative Care

Constant supervision	Suction	Prevention of crusting and tracheitis	Tracheostomy tube care
<ul style="list-style-type: none"> ♦ For bleeding, displacement of tube, blocking of tube and removal of secretions ♦ Temperature, pulse and BP recorded 4 hourly 	<ul style="list-style-type: none"> ♦ Every half hourly to hourly depending upon amount of secretion ♦ Done by applying suction to catheter only when withdrawing it 	<ul style="list-style-type: none"> ♦ Proper humidification by use of humidifier, steam tent, ultrasonic nebulizer or keeping a boiling kettle in room ♦ Loosening crusts using few drops of normal or hypotonic saline or Ringer's lactate every 2–3 hours along with acetylcysteine to liquefy tenacious secretions 	<ul style="list-style-type: none"> ♦ Inner cannula should be removed and cleaned, as and when indicated for first 3 days ♦ Outer cannula should not be removed for 3–4 days unless indicated (for allowing track to be formed) after which it should be removed and cleaned every day

Decannulation

- Decannulation is removal of tracheal tube which should be done as early as possible.

Procedure	Decannulation problems	Causes of decannulation problems
<ul style="list-style-type: none"> Plug tracheostomy tube and observe whether patient can tolerate it for 24 hours If patient can tolerate it, then tube can be safely removed (in operation theatre if feasible) Wound is taped and patient is closely observed Wound healing takes few days to week 	(seen commonly in infants and children) <ul style="list-style-type: none"> Respiratory distress Tachycardia Cyanosis 	<ul style="list-style-type: none"> Persistence of underlying conditions Obstructing granulations around stoma or below it Tracheal edema or subglottic stenosis Incurving of tracheal wall at site of tracheostome Tracheomalacia In-coordination of laryngeal opening reflex Inability to tolerate upper airway resistance Psychological dependence on tracheostomy

Complications

Immediate (on operating table)	Intermediate (few hours to days)	Late (due to prolonged use of tube for weeks and months)
<ul style="list-style-type: none"> Hemorrhage Apnea (in patients with prolonged respiratory obstruction due to sudden washing out of CO₂) Pneumothorax (due to injury to apical pleura) Injury to recurrent laryngeal nerves Aspiration of blood Injury to esophagus (resulting in tracheoesophageal fistula) Injury to high innominate artery (in children) 	<ul style="list-style-type: none"> Reactionary hemorrhage (within 48 hours) Secondary bleeding (on 5th to 8th day due to infection) Displacement of tube Blocking of tube Subcutaneous emphysema Tracheitis and tracheobronchitis with crusting in trachea Atelectasis Lung abscess Local wound infection and granulations 	<ul style="list-style-type: none"> Hemorrhage (due to erosion of major vessel) Laryngeal stenosis (due to perichondritis of cricoid cartilage) Tracheal stenosis (due to tracheal ulceration and infection) Tracheomalacia (due to excision of large area of trachea) Tracheoesophageal fistula (due to prolonged use of cuffed tube or erosion of trachea by tip of tube) Decannulation problems (seen commonly in infants and children) Persistent tracheocutaneous fistula Tracheostomy scar or keloid Corrosion of tracheostomy tube and aspiration of its fragments into tracheobronchial tree

Sequel	Functions	Advantages (over prolonged endotracheal intubation) ¹
<ul style="list-style-type: none"> Loss of speech (because of bypassing of larynx) Anosmia (because patient does not breath through nose) 	<ul style="list-style-type: none"> Alternative pathway for breathing Improves alveolar ventilation (↓ dead space by 30–50% and reducing airflow resistance) Protects airway against aspiration of pharyngeal secretions, blood, etc. Permits removal of tracheobronchial secretions (in patients unable to cough) Permits prolonged intermittent positive pressure respiration (more than 72 hours) Permits administration of anesthesia, medications or humidification 	<ul style="list-style-type: none"> Reduces patient discomfort Reduces need for sedation Improves ability to maintain oral and bronchial hygiene Reduces risk of glottic trauma Reduces dead space and reduces work of breathing Augments process of weaning from ventilatory support

Ref:

- <http://www.surgical-tutor.org.uk/default-home.htm?system/hnep/tracheostomy.htm>~right accessed on 25th Dec. 2009.

10. Antral lavage.

- Antral lavage is a surgical process of irrigating maxillary sinus by puncturing medial wall of maxillary sinus and irrigating sinus with water which flows out through ostium along with exudate
- Also called proof puncture as it helps to prove diagnosis of maxillary pathology based on characteristics of returning fluid.

Indications		Contraindications
Diagnostic (proof puncture)	Therapeutic	
<ul style="list-style-type: none"> ♦ To confirm diagnosis of chronic and subacute maxillary sinusitis evident by mucopurulent or purulent returning fluid ♦ To collect antral content for culture and sensitivity ♦ To collect antral content for exfoliative cytology to confirm malignancy 	<ul style="list-style-type: none"> ♦ Chronic and subacute maxillary sinusitis to drain out pus ♦ Oro-antral fistula associated with sinusitis 	<ul style="list-style-type: none"> ♦ Children under 7–8 years (more chances of false passage due to small sinus size) ♦ Acute maxillary sinusitis (may cause osteomyelitis) ♦ Fracture of maxilla (may cause leakage of fluid along fracture lines) ♦ Bleeding disorders ♦ General disorders like diabetes, hypertension

Anesthesia

- Local (in adults) by placing 4% lignocaine with adrenaline pack in inferior meatus for 10–15 min
- General (in children).

Position

- Sitting or semi-sitting position (for local anesthesia)
- Tonsillectomy position (during general anesthesia).

Procedure

i. Insertion

- Insert Lichwitz trocar and cannula in inferior meatus and pass backwards and upwards directed towards homolateral ear till it is arrested by peak of a hillock like attachment of inferior turbinate, about 2 cm behind anterior end of turbinate.

ii. Puncturing

- Puncture site is located at point 1.5–2 cm from anterior end of inferior turbinate and near attachment of concha with lateral wall as bone is very thin here and can be easily pierced
- Direct trocar and cannula backwards and laterally towards outer cantus of eye and by screwing motion, pierce nasoantral wall till it cracks (sudden loss of resistance) and then remove trocar
- Advance cannula till it reaches opposite antral wall and then withdraw it little.

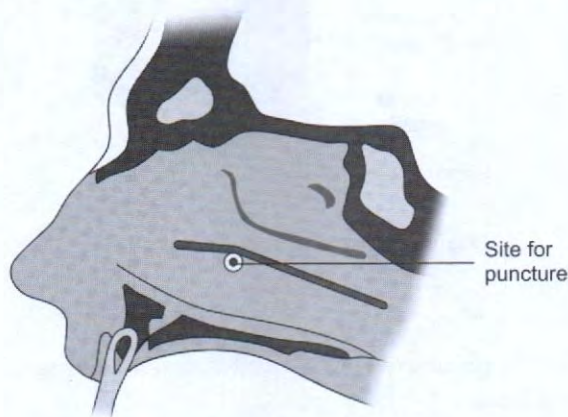


Figure 9: Antral lavage—site for puncture

iii. Lavage

- Bend patient's head downwards and forwards and ask him to breath through mouth
- Irrigate antrum with normal saline at 37°C using 20 mL syringe or Hagginson's syringe
- Continue syringing till return is clear
- Remove cannula and pack inferior meatus to control bleeding.

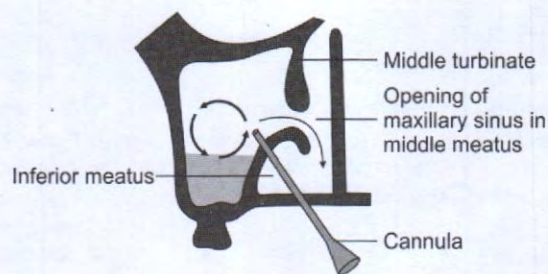


Figure 10: Antral lavage

Postoperative Care

- Removal nasal pack after an hour
- Antibiotic administration for 5–6 days if infected
- Nasal decongestant drops to improve ostium patency
- Analgesics for headache or postoperative pain if any.

Complications

i. Vasovagal syncope	♦ Due to overstimulation of vagus, causing patient to become pale, faint or fall down with slow pulse
ii. Bleeding	♦ Due to injury to nasal mucosa ♦ Secondary or reactionary hemorrhage is rare
iii. False passage due to faulty technique	♦ Anteriorly, trocar may enter cheek instead of antrum resulting in swelling of cheek ♦ Posteriorly, puncture of posterior antral wall causes swelling of posterior part of cheek ♦ If roof of antrum is pierced, may result in orbital injury and cellulitis
iv. Air embolism	♦ Rare but fatal ♦ Prevented by avoiding insufflations of air into antrum after lavage
v. Infection	♦ May be introduced through saline or instrument
vi. Anesthetic complications	

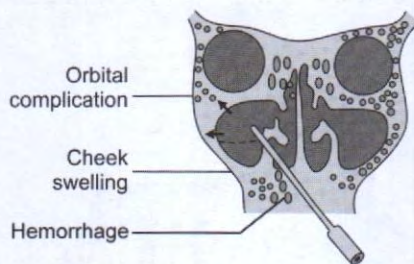


Figure 11: Antral lavage—complications

11. Papillomas of larynx.

- Papillomas are fronds of connective tissue covered by well-differentiated squamous epithelial covering with no invasion of stroma or submucosal tissues
- They are most neoplasm of larynx.

Types

- Juvenile type
- Adult onset type

	<i>Juvenile type</i>	<i>Adult onset type</i>
	<ul style="list-style-type: none"> ♦ Recurrent laryngeal papilloma is squamous papilloma of larynx occurring in infants and young children ♦ So called because of its aggressive behavior and tendency to recur even after surgical removal ♦ Also called juvenile papilloma 	<ul style="list-style-type: none"> ♦ Also called solitary laryngeal papilloma
Etiology	<p><i>Causative organism</i></p> <ul style="list-style-type: none"> ♦ Human papillomavirus—type 6 and 11 <p><i>Source of infection</i></p> <ul style="list-style-type: none"> ♦ Infected mothers genitals during delivery 	<ul style="list-style-type: none"> ♦ Human papillomavirus
Pathology	<p><i>Gross appearance</i></p> <ul style="list-style-type: none"> ♦ Glistening white warty irregular mass <p><i>Microscopic examination</i></p> <ul style="list-style-type: none"> ♦ Finger like projection of epithelial tumor cells with central fibrovascular core 	—
Clinical features	<ul style="list-style-type: none"> ♦ Commonly affects infants and young children 	<ul style="list-style-type: none"> ♦ Common in males aged between 30–50 years
Symptom	<ul style="list-style-type: none"> ♦ Hoarseness ♦ Difficulty in crying ♦ Stridor 	<ul style="list-style-type: none"> ♦ Hoarseness of voice ♦ Dyspnea, stridor or intermittent choking (in case of large papilloma)
Signs	<ul style="list-style-type: none"> ♦ Glistening white irregular growths on true and false cords and epiglottis ♦ Multiple ♦ May be sessile or pedunculated ♦ Friable and bleeds easily <div data-bbox="517 983 734 1205" data-label="Image"> </div> <p style="text-align: center;">Figure 12: Juvenile laryngeal papillomas</p>	<ul style="list-style-type: none"> ♦ Single, small, white to pinkish red, glistening mulberry like polypoid mass ♦ Bleeds on touching ♦ Arises from free edge of anterior half of vocal cord or anterior commissure ♦ May be sessile or pedunculated
Treatment		
a. Conservative	<ul style="list-style-type: none"> ♦ Interferon therapy (to prevent recurrence after surgical removal) 	
b. Operative	<ul style="list-style-type: none"> ♦ Endoscopic removal (using cup forceps, cryotherapy, CO₂ laser, microelectrocautery) 	<ul style="list-style-type: none"> ♦ Excision through microlaryngosurgery ♦ Excision using KTP-532 or CO₂ laser
Prognosis	<ul style="list-style-type: none"> ♦ Recurrence quite common even after surgical removal ♦ Disappear spontaneously after puberty 	<ul style="list-style-type: none"> ♦ Premalignant condition but less aggressive ♦ No recurrence after excision

12. Functional endoscopic sinus surgery (FESS).

- FESS is a recent advance in sinus surgery with promising future as it causes minimal damage to mucosa and reverses pathological changes by ensuring proper drainage and ventilation of sinuses.

Principle

- FESS is based on normal mucociliary clearance pathway wherein it clears obstruction to drainage of ostia to help clearance of mucoid secretion.

Indications		Contraindications
Diagnostic	Therapeutic	
<ul style="list-style-type: none"> ♦ To visualize and evaluate inaccessible areas in the nasopharynx and nasal cavity like middle and superior meatus ♦ Precise visualization and diagnosis of the cause of sinusitis, nasal obstruction, anosmia and headache ♦ Evaluate epistaxis and CSF rhinorrhea ♦ Early diagnosis, biopsy, follow up of nasal, paranasal and nasopharyngeal tumors 	<ul style="list-style-type: none"> ♦ Chronic sinusitis (refractory to conservative treatment) ♦ Sinonasal polypectomy ♦ Mucocele drainage ♦ Foreign body removal ♦ Cauterization in case of epistaxis ♦ Choanal atresia ♦ CSF rhinorrhea ♦ Extramucosal fungal sinusitis ♦ Intranasal dacryocystorhinostomy ♦ Transnasal hypophysectomy 	<ul style="list-style-type: none"> ♦ Gross deviated nasal septum ♦ Acute infections ♦ Inaccessible pathology (lateral frontal sinus disease, stenosis of internal opening of frontal sinus) ♦ Osteomyelitis ♦ Threatened intracranial or intraorbital complication ♦ General conditions like hypertension, diabetes, bleeding disorders, etc.

Anesthesia

Local anesthesia (preferred)	General anesthesia
By packing nose with cottonoid strips soaked in 4% lignocaine with adrenaline followed by injection of 2% lignocaine with adrenaline into uncinate process, bulla ethmoidalis and middle turbinate	In children and uncooperative patients

Position

- Supine position with head resting on a ring or head rest with 15° elevation.

Procedure

a. Diagnostic

1st pass	2nd pass	3rd pass
Pass 0 degree or 30 degree endoscope between nasal septum and inferior turbinate along floor of nasal cavity to visualize whole area up to choana, visualizing both Eustachian tube and nasopharynx	Pass endoscope along superior surface of inferior turbinate to examine any pathology in middle meatus	Pass endoscope between middle turbinate and septum up to anterior wall of sphenoid sinus and its ostium

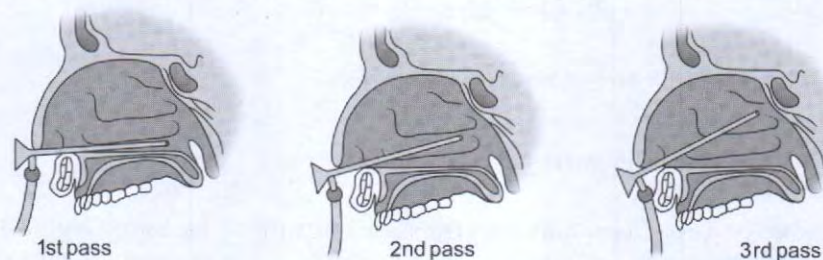


Figure 13: Diagnostic nasal endoscopy

b. Therapeutic (steps of operation)

i. Uncinectomy (infundibulotomy)

- Incise circumferentially over mucosa following course of uncinate process immediately anterior to uncinate process till just above inferior turbinate
- Direct knife inferiorly and parallel to lateral nasal wall to prevent injury to lamina papyracea
- Using straight perichondrial elevator, sublunate uncinate process medially from its superior to inferior attachment
- Remove sublunate uncinate process thus opening infundibulum and exposing bulla ethmoidalis.

ii. Anterior ethmoidectomy

- Open bulla ethmoidalis antero-infero-medially by gently pushing forces or suction tip
- Now resect entire bulla ethmoidalis step by step followed by removal of anterior and middle cells to bulla and sinus lateralis
- Dissection is limited to ethmoidal roof superiorly to avoid injury to anterior ethmoidal artery and dura.

- iii. Middle meatal antrostomy
 - Enlarge maxillary sinus ostium towards anterior frontenelle using back biting forceps if osteium is stenosed
 - Connect natural and accessory ostium if accessory ostium is present.
- iv. Exploration of frontal recess
 - 30 degree or 45 degree endoscope is used to explore frontal recess to clear any pathology
 - Frontal sinus can be opened after removal of cranial part of uncinate process.
- v. Posterior ethmoidectomy and sphenoidotomy
 - Posterior ethmoid is entered by opening ground lamella inferomedially to exenterate posterior ethmoidal cells if diseased
 - Sphenoid is entered by opening bulge of sphenoid sinus inferomedially under direct vision (to avoid any damage to optic nerve and internal carotid artery).
- vi. Nasal packs
 - Apply nasal packs to stop any bleeding from nose.

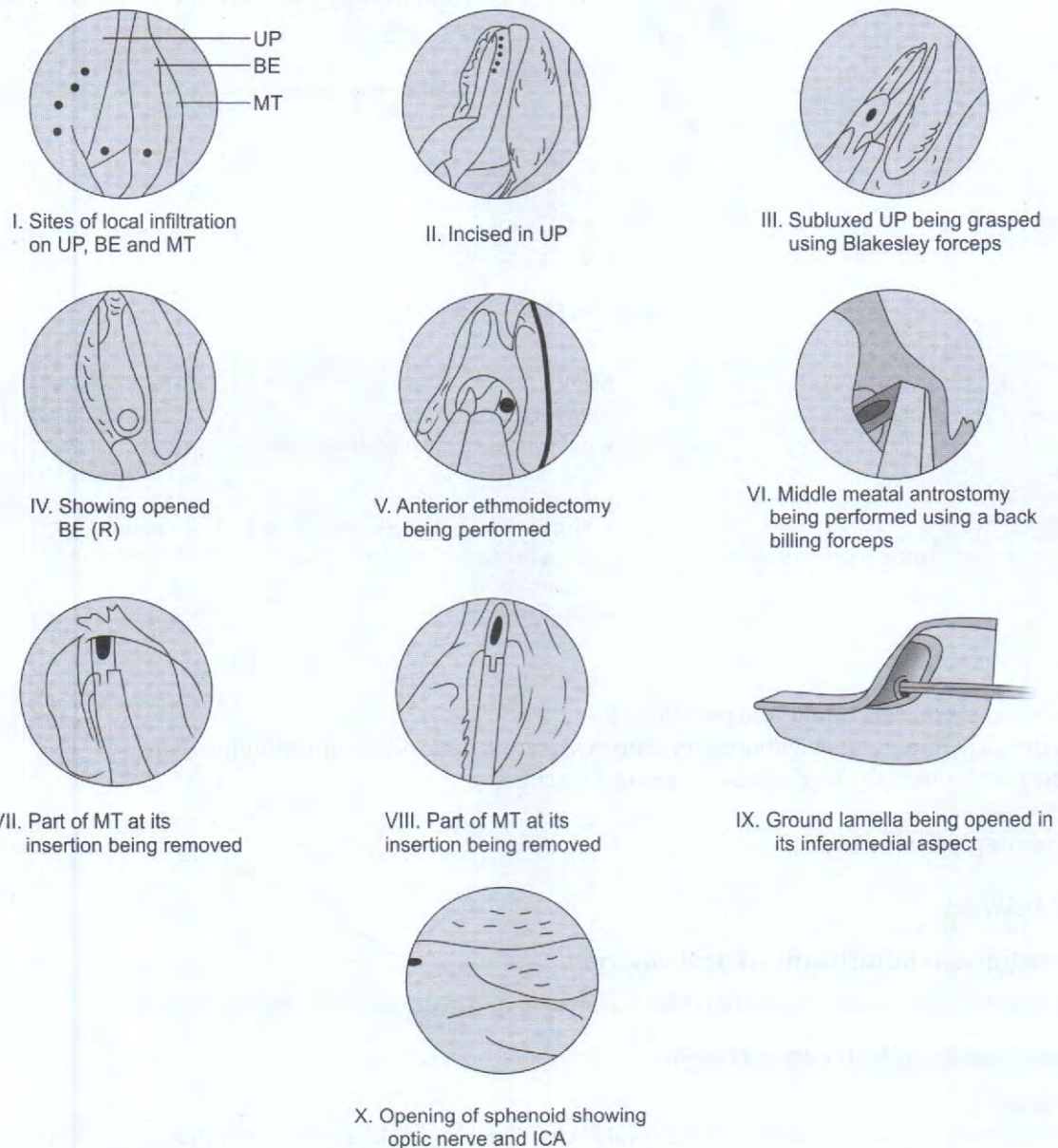
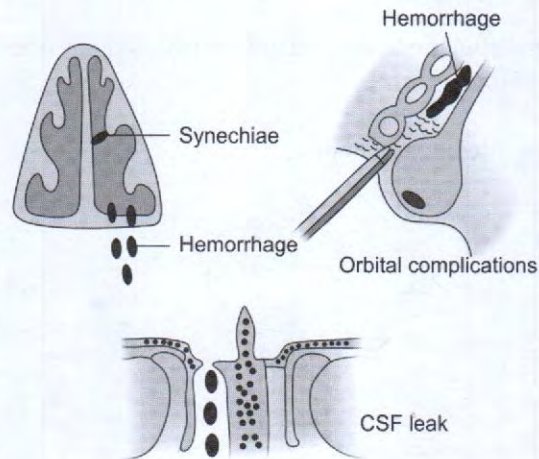


Figure 14: FESS—steps of operation

Postoperative Care

Removal of nasal packs	Cleaning of noses	Antibiotics	Analgesics
After 24 hours	On 2nd day followed by biweekly for 6 weeks (as impaired mucociliary mechanism takes approximately 6 weeks to become functional)	Started during operation and continued for 7–10 days post-operatively	To relieve pain

Complications**Figure 15: FESS—complications**

Major	Minor	Rare
<ul style="list-style-type: none"> ♦ Hemorrhage (orbital/intracranial) ♦ Blindness ♦ Diplopia ♦ CSF rhinorrhea ♦ Anosmia ♦ Injury to important structures (internal carotid artery) ♦ Epiphora 	<ul style="list-style-type: none"> ♦ Periorbital ecchymosis ♦ Subcutaneous and orbital emphysema ♦ Postoperative epistaxis ♦ Postoperative infection ♦ Postoperative adhesions in nasal cavity ♦ Stenosis of maxillary or frontal sinus ♦ Hyposmia ♦ Toothache 	<ul style="list-style-type: none"> ♦ Cavernous-internal carotid artery fistula ♦ Pneumocephalus ♦ Meningitis, brain abscess ♦ Death

Advantages

- Better visualization of anatomy and pathology
- Less damage to important neighboring structures like orbit, optic nerve and cribriform plate
- Endoscopic photography for documentation and teaching
- Cosmetically better
- Minimal hospitalization.

SHORT ANSWERS**13. Premalignant conditions of oral cavity.**

- Premalignant conditions are lesion which have potential to turn malignant if not managed promptly

Premalignant Conditions of Oral Cavity and Pharynx**a. Leukoplakia**

- Leukoplakia is a clinical white patch in oral cavity that cannot characterized clinically or pathologically as any other disease (WHO).

Etiology	Sites involved	Pathology (microscopic appearance)
<ul style="list-style-type: none"> ♦ Smoking ♦ Tobacco chewing ♦ Alcohol abuse (particularly if combined with alcohol abuse) ♦ Chronic trauma from ill fitting dentures or cheek bites ♦ Spicy food ♦ Syphilis ♦ Submucous fibrosis ♦ Hyperplastic candidiasis ♦ Plummer Vinson syndrome 	<ul style="list-style-type: none"> ♦ Buccal mucosa (most common) ♦ Oral commissures ♦ Floor of mouth ♦ Tongue ♦ Gingivobuccal sulcus ♦ Mucosal surface of lip 	<ul style="list-style-type: none"> ♦ Mild to severe epithelial dysplasia ♦ Degree of dysplasia related to malignant potential ♦ Hyperkeratosis and parakeratosis with widening of rete pegs

Clinical features

- Mostly seen in 4th decade of life
- Males affected 2–3 times more compared to females.

Symptoms	Signs
<ul style="list-style-type: none"> ♦ White keratotic plaque in mouth which cannot be rubbed off 	<ul style="list-style-type: none"> ♦ Smooth or wrinkled white patch (homogenous type) ♦ White patches or nodules on erythematous base (nodular or speckled type) ♦ Leukoplakia (white patch) interspersed with erythroplakia along with erosions and fissures (Erosive type or erythroleukoplakia)

Investigations

- Biopsy to rule out malignancy.

Treatment

Supportive	Specific
<ul style="list-style-type: none"> ♦ Spontaneous regression if causative agent is removed 	<ul style="list-style-type: none"> ♦ Surgical excision or ablation with laser or cryotherapy for suspicious small lesions

Differential diagnosis	Prognosis (malignant potential)
<ul style="list-style-type: none"> ♦ Lichen planus ♦ Discoid lupus erythematosus ♦ White spongy nevus ♦ Candidiasis 	<ul style="list-style-type: none"> ♦ Average 5% of leukoplakia (1–17.5%) turn malignant ♦ Malignant potential depends upon site and type of leukoplakia and duration of follow up ♦ Nodular and erosive types are more associated with malignancy

b. Erythroplakia

- Erythroplakia is red patch or plaque on oral mucosal surface
- Red color is due to decreased keratinization and resulting in shining through of underlying red vascular connective tissue of submucosa.

Sites involved	Pathology (microscopic appearance)	Types
<ul style="list-style-type: none"> ♦ Lower alveolar mucosa ♦ Gingivobuccal sulcus ♦ Floor of mouth 	<ul style="list-style-type: none"> ♦ Erythroplakia exhibits severe dysplasia, carcinoma in situ or frank invasive carcinoma 	<ul style="list-style-type: none"> ♦ Homogenous type ♦ Speckled or granular type ♦ Erythroplakia interspersed with area of leukoplakia

Treatment	Prognosis (malignant potential)
<ul style="list-style-type: none"> ♦ Excision biopsy ♦ Follow up 	<ul style="list-style-type: none"> ♦ Malignant potential of erythroplakia is 17 times higher than leukoplakia

c. Melanosis and mucosal hyperpigmentation

- Benign pigmented lesions of oral mucosa may transform into malignant melanoma
- Lesion may resemble benign lesions
- Diagnosed by biopsy.

d. Submucous fibrosis

- Tropical disease occurring mainly in Indian subcontinent (India, Pakistan and Bangladesh).

<i>Etiology</i>	<i>Predisposing factors</i>
♦ Chronic irritation of oral mucosa by betel nuts, spicy foods	i. Irritants <ul style="list-style-type: none"> – Chewing of paan, tobacco, betel nuts – Rubbing of misri on gums and teeth – Chillies and spicy food ii. Trauma <ul style="list-style-type: none"> – Repeated mechanical and thermal trauma iii. Nutrition <ul style="list-style-type: none"> – Poor nourishment may contribute – Associated with vitamin deficiencies iv. Poor dental hygiene

Pathology

<i>Gross appearance</i>	<i>Microscopic appearance</i>
♦ Diffuse, dense white patch behind last molars and in front of ramus of mandible	♦ Deposition of fibrous and hyaline tissue in submucosa of oral cavity or oropharynx

Clinical features

- Usually affects between 15–40 years of age
- Females are more likely to be affected.

<i>Symptoms</i>	<i>Signs</i>
♦ Difficulty in chewing ♦ Burning sensation in oral cavity ♦ Recurrent ulcers of oral cavity ♦ Difficulty in blowing, whistling or sucking due to involvement of cheek ♦ Swelling in neck due to enlargement of lymph nodes	♦ Diffuse and dense white patches in oral cavity on cheeks, retromolar area, hard and soft palate and oropharynx ♦ Small and contracted uvula ♦ Trismus of mild to severe degree ♦ Vesicle formation leading to painful ulceration in early stages ♦ Lymphadenitis in submandibular region

<i>Investigations</i>	<i>Differential diagnosis</i>
i. Hemogram <ul style="list-style-type: none"> – May reveal anemia ii. Gastric analysis <ul style="list-style-type: none"> – May reveal achlorhydria or hyperacidity iii. Biopsy <ul style="list-style-type: none"> – If malignant change is suspected 	i. Trismus ii. Leukoplakia <ul style="list-style-type: none"> – Thickened mucosa with cracks on patches affecting cheeks, lips and tongue iii. Lichen planus <ul style="list-style-type: none"> – Thin white submucous fibrous bands which disappear and reappear – Also involves skin iv. Trush <ul style="list-style-type: none"> – Candidial infection of oral cavity manifesting as curd like plaques

Treatment

<i>Supportive</i>	<i>Specific</i>	
	<i>Conservative</i>	<i>Operative</i>
♦ Removal of irritants (avoiding pan, tobacco, betel nut chewing) ♦ Physiotherapy for trismus ♦ Antacids for hyperacidity ♦ Antioxidants to prevent dysplastic change ♦ Correction of dental hygiene ♦ Improvement in nutrition	i. Hydrocortisone <ul style="list-style-type: none"> – 0.5 cc injection of hydrocortisone submucosally on either side once a week for a month – May reduce trismus ii. Hyaluronidase <ul style="list-style-type: none"> – Injected submucosally to reduced trismus 	i. Excision and grafting <ul style="list-style-type: none"> – Divide fibrous bands in retromolar area and forcibly open mouth – Graft raw area by split skin grafts or rotating lingual mucosal flap ii. Laser surgery (KTP-532 or CO ₂) <ul style="list-style-type: none"> – Useful dividing fibrous bands iii. Condylectomy <ul style="list-style-type: none"> – In advanced cases with severe trismus

e. Lichen planus

- Premalignant potential in dispute
- Common lesion of oral cavity which can undergo occasional epithelial dysplasia or carcinoma
- Lesion resembles white lace like pattern on oral mucosa
- Biopsy mandatory for diagnosis and assess prognosis.

14. Quinsy.

Refer Question No. 7 June 2011 (RS2).

15. Esophageal perforations.

- An emergency condition due to tear in esophageal wall.

<i>Etiology</i>	<i>Sites of perforation (types)</i>
<ul style="list-style-type: none"> ♦ Ingestion of sharp pointed foreign bodies <ul style="list-style-type: none"> – Metallic pins, pointed dentures, fish or meat bones ♦ Instrumental trauma <ul style="list-style-type: none"> – During esophagoscopy, foreign body removal or dilatation of strictures with bougies ♦ Spontaneous <ul style="list-style-type: none"> – Usually follows vomiting 	<ul style="list-style-type: none"> ♦ Just above upper sphincter (during instrumental trauma)—cervical ♦ Lower third esophagus (spontaneous rupture)—thoracic

Clinical Features

<i>Symptoms</i>	<i>Signs</i>
<ul style="list-style-type: none"> ♦ History of esophagoscopy ♦ Pain in neck, chest, epigastrium referred to interscapular region ♦ Fever 	<ul style="list-style-type: none"> ♦ Local tenderness ♦ Surgical emphysema in neck ♦ Crunching sound over heart—Hamman's sign (due to air in mediastinum and pneumothorax) ♦ Signs of shock like fall in BP and raised pulse rate

Investigations

- X-ray chest and neck
 - Widening of mediastinum and retrovisceral space
 - Surgical emphysema
 - Pneumothorax
 - Pleural effusion
 - Gas under diaphragm.

Treatment

	<i>Conservative</i>	<i>Operative</i>
Indications	<ul style="list-style-type: none"> ♦ Early perforations 	<ul style="list-style-type: none"> ♦ Progressive surgical emphysema ♦ Evidence of pleural effusion ♦ Worsening of patients general condition ♦ Formation of mediastinal abscess
Technique	<ul style="list-style-type: none"> ♦ Stop all oral feeds immediately ♦ Parenteral nutrition ♦ Systemic antibiotics intravenously in massive doses ♦ Steroids in large doses 	<ul style="list-style-type: none"> ♦ Repair of perforation ♦ Drainage of retrovisceral space and/or upper mediastinum if suppuration develops

16. Myringotomy.

Refer Question No. 1 December 2012 (RS2).

17. Nasal myiasis.

- Nasal myiasis is infestation of nose, nasopharynx and paranasal sinuses by maggots (larval form of flies).

Etiopathogenesis

Causative organism	Predisposing factors	Pathogenesis
Fly (<i>Chrysomia benziana</i>)	<ul style="list-style-type: none"> Foul smelling nasal discharge due to <ul style="list-style-type: none"> Atrophic rhinitis Suppurative sinusitis Syphilis Leprosy Infected wound Loss of nasal sensation Poor nasal hygiene Comatose patient Uncontrolled diabetes Immunocompromised states Malignancy of maxilla Osteoradionecrosis following radiotherapy 	Foul smelling discharge → attraction of flies → laying of about 200 eggs at a time → hatching of eggs within 24 hours → formation of larvae → penetration into surrounding tissue → secretion of proteolytic enzymes → extensive tissue destruction

Pathology

- High concentration infiltration by lymphocytes, giant cells, neutrophils, eosinophils and plasma cells.

Clinical Features

Symptoms		Signs
Initially (first 3–4 days)	Later (after 3–4 days)	
<ul style="list-style-type: none"> Intense itching and sneezing due to movements of maggots Lacrimation Headache Nasal obstruction due to inflamed mucosa Blood stained discharge due to invasion and destruction of surrounding tissue 	<ul style="list-style-type: none"> Crawling of maggots from nose Foul smelling discharge 	<ul style="list-style-type: none"> Congested and edematous nasal mucosa Puffiness of eyelids and lips Cellulitis of nose and face Extensive destruction of nose, sinuses, soft tissue of face, palate and eyeball Fistulae in palate or around nose

Treatment

Conservative		Operative
Supportive	Specific	
<ul style="list-style-type: none"> Nasal hygiene Isolation of patient with mosquito net (to avoid contact with flies) Treatment of underlying cause 	<ul style="list-style-type: none"> Picking up of all visible maggots Instillation of liquid paraffin, diluted chloroform or ether to kill them Nasal packing with turpentine pack to irritate maggots. Alkaline nasal douche to remove slough crusts and dead maggots 	<ul style="list-style-type: none"> Endoscopic intranasal turbinectomy of necrotic turbinates

Complications

- Meningitis
- Death.

18. Tuning fork tests.

- Tuning forks tests are tests for qualitative assessment of hearing
- Commonly performed with tuning fork of frequency 256, 512 and 1024 Hz.

Tuning Fork Tests

Rinne's test	Weber's test	Absolute bone conduction test	Schwabach's test	Bing test	Gelle's test
Compares AC with BC	Detects better hearing cochlea	Measures cochlear function and investigates possible ↓ in BC	Measures cochlear function	Examines effect of ear occlusion on BC	Assess effect of air pressure in ear on BC

a. Rinne's test

- Tuning fork test to compare air conduction of ear with its bone conduction
- Described by Heinrich Rinne.

Objective

- To compare air conduction with bone conduction.

Principle

- Hearing through air conduction is louder and heard twice as long as through bone conduction.

Procedure

- Place base of vibrating tuning fork on mastoid process, until subject no longer feels vibrations (bone conduction)
- Then transfer tuning fork to front of same side ear about 1 cm away from external ear (air conduction)
- Alternatively, ask patient to compare loudness of sound heard by air conduction and bone conduction.

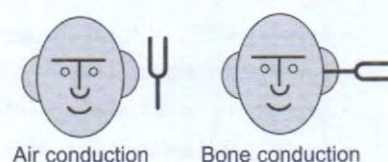


Figure 16: Rinne's test

Inference

Tuning fork results	Rinne's nomenclature	Observation	Diagnosis
♦ AC > BC	R+ (Rinne's positive)	Vibrations heard in air even after cessation of bone conduction	Normal hearing
♦ AC > BC (both reduced)	R+ (reduced)	Vibrations heard in air even after cessation of bone conduction but reduced compared to other ear	Sensorineural deafness
♦ AC = BC	R± (Rinne's equivocal)	Time of hearing vibration in air equals time of hearing vibration on bone	Slight conductive deafness
♦ AC only, no BC	R+ infinitely	Vibration heard in air only but not on bone	Severe sensorineural deafness
♦ BC > AC	R-	Vibrations in air are not heard after cessation of bone conduction	Conductive deafness (indicates minimum air—bone gap of 15–20 dB)
♦ BC only, with untested ear masked	R- infinitely	Vibrations felt on bone only but not in air	Very severe conductive deafness
♦ BC only, with no masking of untested ear	R false (False negative Rinne's)	Vibrations in air are not perceived but bone conduction is positive (due to transcranial transmission of sound from opposite ear) Correct diagnosis can be made by masking non-test ear with Barany's noise box while testing for bone conduction	Very severe or total sensorineural deafness

Application

- To find out type of deafness either conduction or sensorineural deafness
- To predict bone air gap

Rinne's interpretation	Air-bone gap
♦ Negative for 256 Hz but positive for 512 Hz	20–30 dB
♦ Negative for 256 Hz and 512 Hz but positive for 1024 Hz	30–45 Hz
♦ Negative for 256 Hz, 512 Hz and 1024 Hz	45–60 Hz

b. Weber's test

- Demonstrated by Ernest Weber who described lateralization of bone conducted sound of an occluded ear
- Also called Chimani moosa test or lateralization test.

Objective

- To detect better hearing cochlea.

Principle

- Air conduction masks bone conduction.

Procedure

- Place base of vibrating tuning fork on midline of head (vertex of skull, forehead, bridge of nose, upper incisor) but base of nose preferred as skin is thinnest.

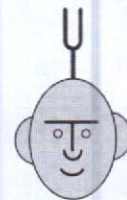


Figure 17: Weber's test

Inference

Observation	Weber's nomenclature	Diagnosis
♦ Sound heard equally on both sides	Weber's centralized	Normal hearing
♦ Sound heard at center of head	Weber's centralized	Bilateral symmetrical conductive deafness
♦ Sound heard louder in diseased ear	Weber's lateralized to worse ear	Conductive deafness
♦ Sound heard louder in normal ear	Weber's lateralized to better ear	Sensorineural deafness

Application

- To diagnose type of deafness either conduction or sensorineural deafness
- To find out affected ear
- To diagnose malingering
- To detect false Rinne's negative
- To assess degree of hearing loss
 - Lateralization of sound in Weber test with a tuning fork of 512 Hz implies a conductive loss of 15–25 dB in ipsilateral ear or a sensorineural loss in contralateral ear.

c. Absolute bone conduction test

- Also called modified Schwabach's test.

Objective

- To measure cochlear function and investigate possible decrease in bone conduction.

Principle

- Bone conduction of patient is compared with that of the examiner assuming it to be normal.

Procedure

- Occlude external auditory meatus of test ear by pressing tragus
- Press base of vibrating tuning fork against mastoid process of patient
- Transfer tuning fork to examiner's ear with tragal occlusion once patient stops hearing.

Inference

Observation	Diagnosis
♦ Patient and examiner hears for same duration	Conductive deafness
♦ Patient hears for shorter duration	Sensorineural deafness
♦ Patient hears for longer duration	Conductive otosclerosis

Applications

- To find out type of deafness
- To assess site of lesion.

d. Schwabach's test

Objective

- To measure cochlear function.

Principle

- Bone conduction of patient is compared with that of examiner presuming it to be normal.

Procedure

- Press base of vibrating tuning fork against mastoid process of patient
- Transfer tuning fork to examiner's ear once patient stops hearing
- However, in this test external auditory meatus is not occluded.

Inference

Observation	Diagnosis
♦ Patient and examiner hears for same duration	Conductive deafness
♦ Patient hears for shorter duration	Sensorineural deafness
♦ Patient hears for longer duration	Conductive otosclerosis

Applications

- To find out type of deafness
- To assess site of lesion.

e. Bing test

Objective

- To examine effect of occlusion of ear canal on bone conduction.

Principle

- Bone conduction is better with inhibition of air conduction.

Procedure

- Press base of vibrating tuning fork against mastoid process of patient
- Alternately, close and open external auditory meatus by pressing tragus inward.

Interference

Interpretation	Observation	Diagnosis
♦ Bing's positive	Hears louder with occluded ear and softer with open ear	Normal hearing Sensorineural deafness
♦ Bing's negative	No change appreciated	Conductive deafness

Application

- To find out type of deafness either conduction or sensorineural deafness.

f. Gelle's test

Objective

- To assess effect of air pressure in external ear on bone conduction.

Principle

- Increased air pressure in external ear pushes tympanic membrane and ossicles inward thus raised intra-labyrinthine pressure and causing immobility of basilar membrane resulting in decreased hearing.

Procedure

- Press base of vibrating tuning fork against mastoid process of patient
- Increase air pressure in ear canal using Siegle's speculum

Interference

<i>Interpretation</i>	<i>Observation</i>	<i>Diagnosis</i>
♦ Gelle's positive	Decrease in hearing	Normal hearing Sensorineural deafness
♦ Gelle's negative	No change appreciated	Conductive deafness

Application

- To find out type of deafness either conduction or sensorineural deafness
- To diagnose stapes fixation in otosclerosis.

19. Septal perforation.

Refer Question No. 12 June 2011 (RS2).

20. Lateral sinus thrombosis.

Refer Question No. 4 June 2009 (RS2).

21. Rhinoscleroma.

Refer Question No. 4 December 2013 (RS2).

22. Membranous conditions of tonsil.

Refer Question No. 12 December 2011 (RS2).

MBBS PHASE III EXAMINATION

JUNE 2008
(Revised Scheme 2)

■ LONG ESSAYS

1. Discuss etiopathology, clinical features and management of carcinoma nasopharynx.

- Nasopharyngeal carcinoma is a multifactorial disease uncommon in India.

Etiology

Causative agent	Predisposing factors
♦ Epstein-Barr virus	<p>a. Genetic susceptibility</p> <ul style="list-style-type: none"> Common in people from southern China, Taiwan, Indonesia, Hong Kong In India, common in people of mongoloid origin, i.e. residents of North East India <p>b. Environmental</p> <ul style="list-style-type: none"> Air pollution Smoke from burning incense and wood <p>c. Diet</p> <ul style="list-style-type: none"> Consumption of dry salted fish (contains nitrosamines) Smoked food <p>d. Personal habits</p> <ul style="list-style-type: none"> Smoking of tobacco and opium

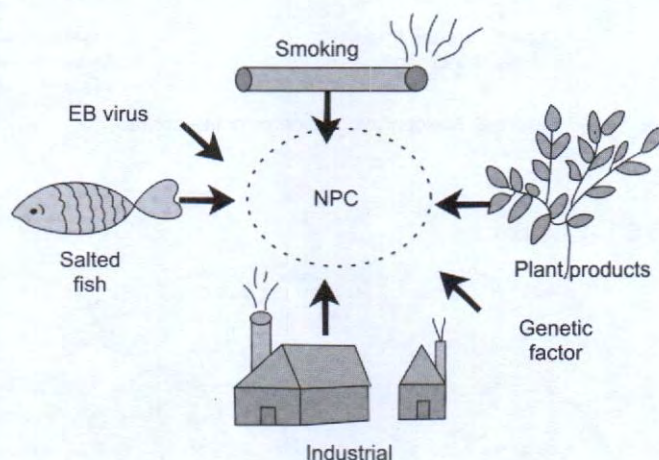


Figure 1: Nasopharyngeal carcinoma—predisposing factors

Pathology (types)

Gross Appearance

- | | |
|------------------------|---------------------------------------|
| i. Proliferative type | Polypoid tumor filling up nasopharynx |
| ii. Ulcerative type | Tumor mass with ulceration |
| iii. Infiltrative type | Tumor invades submucosa |

Histopathology (WHO)

- i. Type I (25%) Squamous cell carcinoma
- ii. Type II (12%) Nonkeratinizing carcinoma (with/without lymphoid stroma)
- iii. Type III (63%) Undifferentiated carcinoma (with/without lymphoid stroma)

Site of Origin

- Fossa of Rosenmuller in lateral wall of nasopharynx (commonest site)
- Posterosuperior wall.

Spread

Direct spread to	Lymphatic spread to	Distant metastases to
<ul style="list-style-type: none">♦ Cranium through foramen lacerum and ovum involving cranial nerves III, IV, V and VI♦ Parapharyngeal space involving cranial nerves IX, X, XI and XII along with pterygoid muscles♦ Eustachian tube, nose and orbit	<ul style="list-style-type: none">♦ Retropharyngeal nodes and cervical nodes	<ul style="list-style-type: none">♦ Liver, lungs and bone (spine)

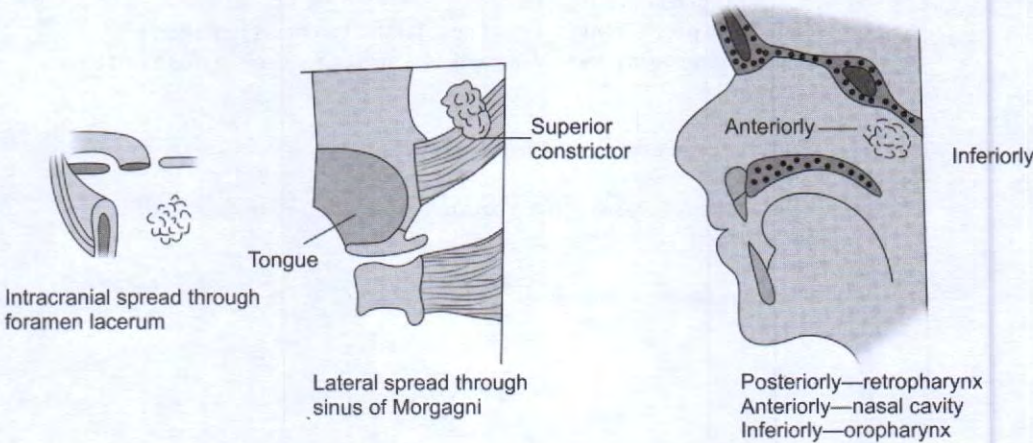


Figure 2: Nasopharyngeal carcinoma—spread

Clinical Features

- Seen mostly in males in their 5th–7th decades

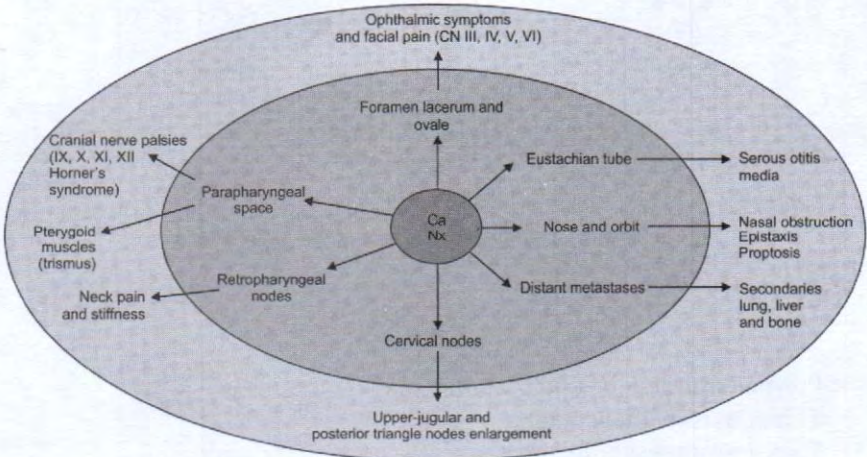


Figure 3: Nasopharyngeal carcinoma—spread and clinical features

Symptoms

Nasal	Otological	Ophthalmoneurological (due to involvement of cranial nerves)	General
<ul style="list-style-type: none"> ♦ Nasal obstruction ♦ Nasal discharge ♦ Denasal speech ♦ Epistaxis 	<ul style="list-style-type: none"> ♦ Ear discharge (mostly unilateral) ♦ Hearing loss ♦ Tinnitus and dizziness 	<ul style="list-style-type: none"> ♦ Diplopia ♦ Headache ♦ Facial and neck pain ♦ Blindness (due involvement of II nerve at apex of orbit) 	<ul style="list-style-type: none"> ♦ Loss of appetite ♦ Weight loss ♦ Lump in neck ♦ Difficulty to open mouth

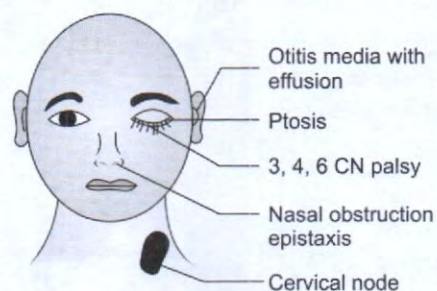


Figure 4: Nasopharyngeal carcinoma—clinical features

Signs

Cervical lymphadenopathy—(Most common)	Nasal	Otological	Ophthalmoneurological
<ul style="list-style-type: none"> ♦ Bilateral enlargement of lymph nodes between angle of jaw and mastoid process and some along spinal accessory in posterior triangle of neck 	<ul style="list-style-type: none"> ♦ Rhinolalia clausa 	<ul style="list-style-type: none"> ♦ Conductive deafness ♦ Serous or suppurative otitis media 	<ul style="list-style-type: none"> ♦ Reduced corneal reflex (due to invasion of V nerve through foramen lacerum) ♦ Squint and ophthalmoplegia (due to involvement of III, IV and VI nerves) ♦ Exophthalmos ♦ Palatal palsy (due to involvement of X nerve) ♦ Trismus (due to involvement of pterygoids) ♦ Jugular foramen syndrome (due to pressure of lateral retropharyngeal lymph nodes or direct involvement of IX, X and XI nerves) ♦ Horner's syndrome (due to involvement of cervical sympathetic chain) ♦ Trotter's triad (triad of conductive deafness, ipsilateral temporoparietal neuralgia and palatal palsy)

Investigations

- Radiology
 - X-rays (skull) and CT scan to demonstrate erosion of bone at base of skull and extent of tumor
 - MRI with gadolinium enhancement to reveal intracranial extension.
- Biopsy
 - Endoscopic or operative (through transpalatal approach).

TNM Staging

Stage	Tumor growth	Nodal involvement	Metastases
X	—	Cannot be assessed	Cannot be assessed
0	—	No regional node metastasis	No distant metastases

Contd...

Contd...

Stage	Tumor growth	Nodal involvement	Metastases
1	Confined to nasopharynx	Unilateral metastasis; above supraclavicular fossa; 6 cm or less	Distant metastasis
2	Extended to oropharynx and/or nasal cavity	Bilateral metastasis; above supraclavicular fossa; 6 cm or less	—
2a	Without parapharyngeal extension		
2b	With parapharyngeal extension		
3	Invasion of bony structures and/or paranasal sinuses	Metastasis in lymph nodes	—
3a		>6 cm	
3b		In supraclavicular fossa	
4	Intracranial extension and/or involvement of cranial nerves, infratemporal fossa, hypopharynx or orbit or masticator space	—	—

Treatment

Chemotherapy	Radiotherapy (treatment of choice)	Operative
Indications <ul style="list-style-type: none"> ♦ In combination with radiotherapy (concomitantly or post-radiotherapy) Regime <ul style="list-style-type: none"> ♦ Cisplatin with/without 5-FU Advantages <ul style="list-style-type: none"> ♦ Doubles cure rate when combined with radiotherapy ♦ Improves local control of tumor ♦ Treats distant metastases 	<ul style="list-style-type: none"> ♦ External or intracavitary implants (brachytherapy) Regime <ul style="list-style-type: none"> ♦ 6000–7000 rads using large ports externally over period of 6 weeks 	<ul style="list-style-type: none"> ♦ Radical neck dissection for persistent nodes ♦ Skull base surgery of recurrent tumor

Prognosis

- Good (>5 years) if diagnosed and treated early.

2. Discuss the physiology of phonation.

- Phonation means production of sound which is a function of larynx acquired during evolution.

Principle

- Phonation is based on Bernoulli's effect which states that during steady flow of a fluid or a gas, pressure is less where velocity is greater
- Thus, when air passes from lungs to pharynx through a constriction, i.e. glottis, velocity of air would be greatest and pressure least at site of constriction.

Mechanism (Aerodynamic Myoelastic Theory)

- When we wish to phonate, recurrent laryngeal nerve sets vocal cords into adducted position, but because vocal processes are slightly bulkier than membranous cord, there exists a slight between membranous cords
- When lungs expel air, airstream passes through this chink between vocal cords
- Then according to Bernoulli's principle, there is drop of pressure at vocal cords causing mucosa of vocal cords to be drawn into gap, eventually blocking it
- This causes rise in subglottic pressure forcing another stream of air flow through cords resulting another pressure drop and closure of gap

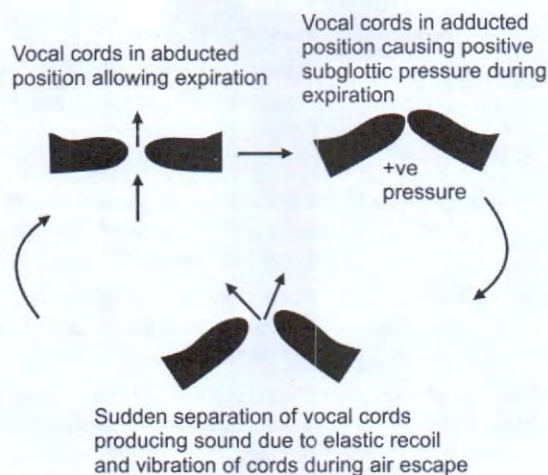


Figure 5: Aerodynamic myoelastic theory

- Repetition of this process develops a vibratory pattern at vocal cords resulting in production of sound
- This sound is modified into speech by tongue, teeth, paranasal sinuses, lip and palate.

Factors Determining Production of Voice

- Pitch is determined by number of vibrations of vocal cord per second depending upon vibratory mass of vocal folds, laryngeal adductors and tensors
- Intensity and duration are determined by capacity of lungs and subglottic pressure build up by adduction of cords
- Quality depends upon resonators and articulators.

SHORT ESSAYS

3. Intrinsic muscles of larynx.

- Larynx is an organ for phonation or voice production and communicates above with pharynx and below with trachea.

Intrinsic Muscles of Larynx

- Muscles which are attached to the laryngeal cartilage.

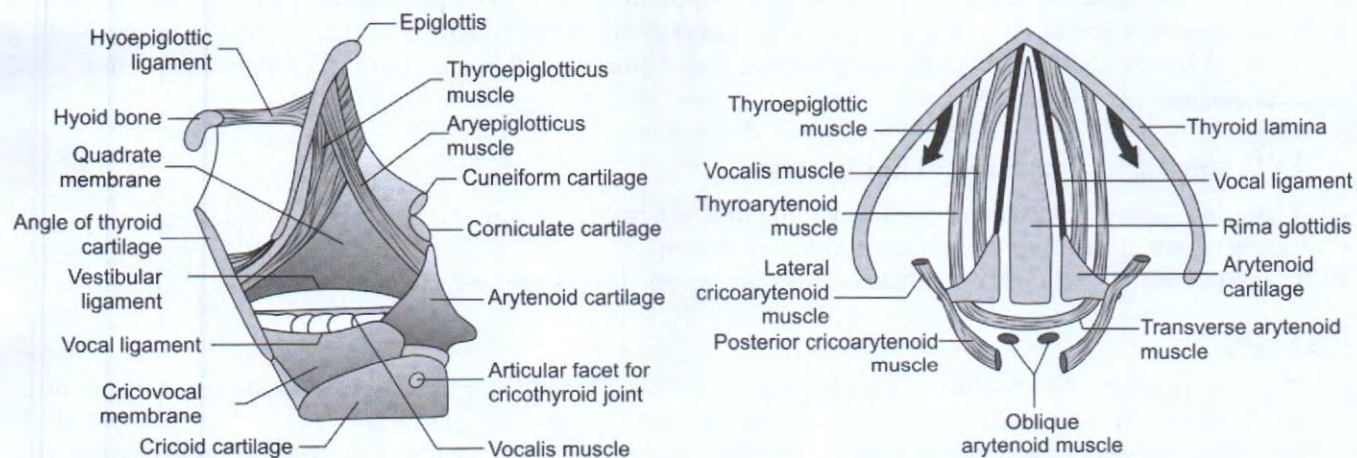


Figure 6: Larynx—intrinsic muscles

Name	Origin	Insertion	Nerve supply	Action
a. Cricothyroid (only intrinsic muscle outside larynx and only muscle supplied by Ext. laryngeal nerve*)	Lower border and lateral surface of cricoid cartilage	Fibers pass backward and upward to insert into inferior cornu and lower border of thyroid cartilage	External laryngeal	<ul style="list-style-type: none"> • Closes glottis • Adducts vocal cords • Tenses vocal cords
b. Posterior cricoarytenoid	Posterior surface of lamina of cricoid cartilage	Fibers pass upward and laterally to get inserted into muscular process of arytenoid cartilage	Recurrent laryngeal	<ul style="list-style-type: none"> • Opens glottis • Abducts vocal cords
c. Lateral cricoarytenoid	Lateral part of upper border of arch of cricoid cartilage	Fibers pass upward and backward to be inserted in to muscular process of arytenoid cartilage	Recurrent laryngeal	<ul style="list-style-type: none"> • Closes glottis • Adducts vocal cords
d. Transverse arytenoids (Only unpaired muscle)	Posterior surface of one arytenoid cartilage	Fibers runs transversely to insert into posterior surface of opposite arytenoid cartilage	Recurrent laryngeal	<ul style="list-style-type: none"> • Closes glottis • Adducts vocal cords
e. Oblique arytenoids	Muscular process of one arytenoid cartilage	Fibers runs obliquely across midline crossing each other to be inserted into apex of opposite arytenoid cartilage	Recurrent laryngeal	<ul style="list-style-type: none"> • Closes inlet of larynx • Relaxes vocal cords
f. Aryepiglotticus	As continuation of oblique arytenoid into aryepiglottic fold	Edge of epiglottic cartilage	Recurrent laryngeal	<ul style="list-style-type: none"> • Closes inlet of larynx
g. Thyroarytenoid	Posterior aspect of angle of thyroid cartilage and adjacent part of cricothyroid ligament	Fibers run backward and upward to reach anterolateral surface of arytenoid cartilage	Recurrent laryngeal	<ul style="list-style-type: none"> • Closes glottis • Adducts vocal cords • Relaxes vocal cords
h. Vocalis	As continuation of thyroarytenoid	Vocal ligament	Recurrent laryngeal	<ul style="list-style-type: none"> • Relaxes vocal cords
i. Thyroepiglotticus	As continuation of thyroarytenoids	Edge of epiglottis	Recurrent laryngeal	<ul style="list-style-type: none"> • Opens inlet of larynx

Applied Anatomy

- Damage to external laryngeal nerve causes some weakness of phonation due to loss of tightening effect of cricothyroid on the vocal cords
- When both the recurrent laryngeal nerves are interrupted, vocal cords lie in cadaveric position and phonation is completely lost. Breathing also becomes difficult
- When only one recurrent laryngeal nerve is paralyzed, opposite vocal cord compensates for it and phonation is possible
- In progressive lesion of recurrent nerve, muscles obeys Semon's law which states that in chronic involvement of recurrent nerves, abductors of larynx are paralyzed first, followed by other muscles and on other hand, in functional paralysis of larynx it is vice versa.

4. TNM staging and its clinical application.

- Staging is a system to determine spread and prognosis of a malignant cancer
- Staging means extent of spread of tumor within the patient
- It can be assessed by clinical examination, investigation and histopathologic examination.

TNM Staging

- TNM staging system for all solid tumors was devised by Pierre Denoix using size and extension of primary tumor, its lymphatic involvement, and presence of metastases to classify progression of cancer
- It was further developed and maintained by Union Internationale Centre Cancer, Geneva (UICC) to achieve consensus on one globally recognised standard for classifying the extent of spread of cancer.

TNM - staging system

Components

- T : extent of the primary tumor
 N : absence or presence and extent of regional lymph Node involvement
 M : absence or presence of distant metastasis

Staging

Numbering	Tumor involvement	Nodal involvement	Metastasis
X	Primary tumor cannot be assessed	Regional lymph nodes cannot be assessed	Distance metastasis cannot be assessed
0	No evidence of primary tumor	Tumor cells absent from regional lymph nodes	No distant metastasis
Is	Carcinoma in situ		
1	Increasing size and/or local extent of the primary tumor	Regional lymph node metastasis present	Metastasis to distant organs
2		Tumor spread to an extent between N1 and N3	—
3		Tumor spread to more distant or numerous regional lymph nodes	—
4		—	—

Types

Clinical staging	Pathological staging	Post therapy staging
<ul style="list-style-type: none"> Staging based on clinical examination Prefixed as c (cTNM) Generally implied in absence of any other prefix 	<ul style="list-style-type: none"> Staging based on examination of pathological specimen Prefixed as p (pTNM) 	<ul style="list-style-type: none"> Staging after neoadjuvant therapy Prefixed as y (yTNM)

Clinical Application

- Aid medical staff in staging tumor helping to plan treatment
- Give an indication of prognosis
- Assist in evaluation of results of treatment
- Enable facilities around the world to collate information more productively
- To contribute to the continuing investigation of human cancer.

Significance

- TNM staging is an ^{Int'l} internally accepted standard in cancer staging used for variety of cancer staging (brain tumor does not TNM staging).

5. Postcricoid malignancy.

- Constitutes 30% of laryngopharyngeal malignancies.

Etiology	Predisposing factors
<ul style="list-style-type: none"> Plummer Vinson syndrome 	<ul style="list-style-type: none"> Betel nut chewing Smoking

Pathology

Gross appearance	Microscopic examination
<ul style="list-style-type: none"> Ulcerative type of lesion 	<ul style="list-style-type: none"> Moderately differentiated squamous cell carcinoma

Spread

- Local spread (annular fashion) involving cervical esophagus, arytenoids or recurrent laryngeal nerve at cricoarytenoid joint
- Lymphatic spread to paratracheal lymph nodes bilaterally.

Clinical Features

- Usually affects females in 3rd and 4th decade of life.

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Progressive dysphagia ♦ Progressive malnutrition ♦ Weight loss ♦ Voice change and aphonia in later stages (due to infiltration of recurrent laryngeal nerve or posterior cricoarytenoid muscle) 	<ul style="list-style-type: none"> ♦ Loss of laryngeal crepitus ♦ Edema and erythema of postcricoid region ♦ Pooling of secretions in hypopharynx

Investigations

Radiography	Endoscopy (direct laryngoscopy)
<ul style="list-style-type: none"> ♦ X-ray (lateral view of neck) shows increased prevertebral shadow ♦ Barium swallow to assess extent of disease 	<ul style="list-style-type: none"> ♦ To assess extent of lesion and take biopsy

Treatment

Radiotherapy	Operative
<p><i>Dose</i></p> <ul style="list-style-type: none"> ♦ 5000–6000 rads of cobalt 60 over 5–6 weeks <p><i>Advantages</i></p> <ul style="list-style-type: none"> ♦ Preserves laryngeal function 	<ul style="list-style-type: none"> ♦ Laryngo-pharyngo-esophagectomy with stomach pull up or colon transposition

Prognosis

- Poor due to early bilateral lymphatic metastases and vertebral column involvement.

6. Lingual thyroid.

- Lingual thyroid is presence of ectopic thyroid tissue in base of tongue.

Etiopathogenesis

- Developmental anomaly in thyroid development
- Thyroid develops from thyroglossal diverticulum
- Lingual thyroid results due to congenital persistence of thyroid tissue in region of foramen cecum (in midline at junction of anterior 2/3rd and posterior 1/3rd of tongue).

Clinical Features

- Usually affects females.

Symptoms	Signs
<ul style="list-style-type: none"> i. Small size <ul style="list-style-type: none"> – Asymptomatic ii. Large (increases in size during period of stress like menarche, hemorrhage into it) <ul style="list-style-type: none"> – Gradually increasing foreign body sensation – Dysphagia – Hot potato speech 	<ul style="list-style-type: none"> ♦ Well demarcated smooth reddish swelling in midline of tongue

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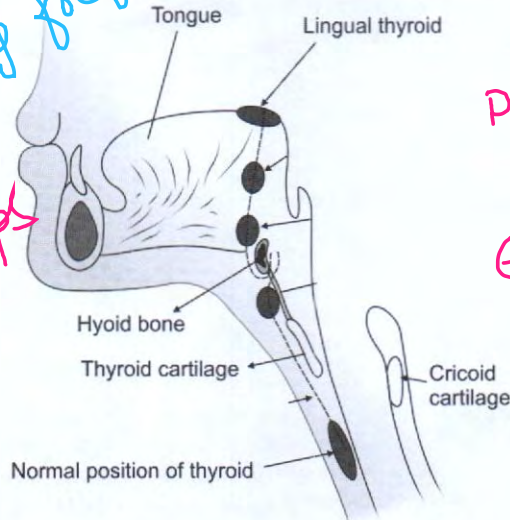


Figure 7: Lingual thyroid

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Investigations

Thyroid function test	Thyroid scan
♦ T3, T4 and TSH assay	♦ Detects ectopic thyroid tissue

Treatment

Conservative	Operative
♦ No treatment if asymptomatic ♦ Thyroxine supplementation if TSH raised	♦ Excision by transoral or transcervical approach (in presence of other functioning thyroid tissue) ♦ Pedicled translocation in lateral cervical gutter or reimplantation in sternomastoid or rectus abdominus muscle (if only functioning thyroid tissue)

7. Etiology of oropharyngeal cancer.

- Oropharyngeal cancers may be benign (papilloma) or malignant (squamous cell carcinoma).

Etiology

Causative agents	Predisposing factors
a. Tobacco <ul style="list-style-type: none"> – Tobacco chewing or smoking accounts for 2/3rd of oropharyngeal cancers – Reverse smoking (keeping burning end of cigarette in mouth) practiced in India also may cause oropharyngeal cancer b. Alcohol <ul style="list-style-type: none"> – People consuming alcohol along with tobacco consumption are at 15 times greater risk – Tobacco and alcohol act synergistically c. Premalignant conditions <ul style="list-style-type: none"> – Premalignant conditions like leukoplakia, erythroplakia or submucous fibrosis may turn malignant 	a. Age <ul style="list-style-type: none"> – Common after 4th decade b. Sex <ul style="list-style-type: none"> – Females are 6 times more prone c. Race <ul style="list-style-type: none"> – Blacks are twice as prone as whites

8. Acute epiglottitis.

- Acute epiglottitis is an acute inflammatory condition of supraglottic region involving epiglottis, aryepiglottic folds and arytenoids
- Also called acute supraglottic laryngitis.

Etiology*Causative Organism*

- Hemophilus influenza type B.

Pathology

- Severe cellulitis of epiglottic and aryepiglottic fold tissue
- Congested and edematous mucosa.

Clinical Features

- Usually affects children aged 2–7 years but can also affect adults
- Abrupt onset with rapid progression.

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Severe fever (40°) due to septicemia ♦ Pain in throat ♦ Dyspnea and stridor (in children) ♦ Sore throat and dysphagia (in adults) ♦ Muffled (hot potato) voice 	<ul style="list-style-type: none"> ♦ Toxic patient with flushed skin, anxious and frightened look ♦ Red and swollen epiglottis (cherry red epiglottis) ♦ Edema and congestion of supraglottic structure ♦ Thick and inspissated secretion ♦ Tripod sign (patient sits up leaning forward supporting on upper limb as inspiratory stridor increases on supine position)

Investigations

Throat swab	X-ray (lateral view of neck)
<ul style="list-style-type: none"> ♦ For culture and sensitivity 	<ul style="list-style-type: none"> ♦ Shows swollen epiglottis (thumb sign) ♦ Narrowed supraglottic airway

Differential Diagnosis

Acute laryngo-tracheo-bronchitis	Laryngeal diphtheria	Laryngeal
<ul style="list-style-type: none"> ♦ Seen in children aged 3 months to 3 years ♦ Slow onset ♦ Low grade or no fever ♦ Barking seal like cough ♦ No difficulty in swallowing ♦ Steeple sign on X-ray (AP view) neck 	<ul style="list-style-type: none"> ♦ Low grade fever ♦ Croupy cough ♦ Grayish white membrane on tonsil, pharynx and soft palate ♦ Bull neck cervical adenopathy 	<ul style="list-style-type: none"> ♦ No cough ♦ Marked respiratory distress ♦ Features of underlying condition

Treatment

Supportive	Conservative	Operative
<ul style="list-style-type: none"> ♦ Hospitalization ♦ Parenteral fluids for adequate hydration ♦ Humidification and oxygen ♦ Steam inhalation and voice rest 	<p><i>Antibiotics (combination therapy)</i></p> <ul style="list-style-type: none"> ♦ Ampicillin/amoxicillin ♦ Chloramphenicol (50 mg/kg/day QID) ♦ Ceftriaxone (75 mg/kg/day BD) ♦ Cefotaxime (50–100 mg/kg/day QID) <p><i>Steroids (to relieve edema)</i></p> <ul style="list-style-type: none"> ♦ Hydrocortisone or dexamethasone parenterally 	<p><i>Tracheostomy</i></p> <ul style="list-style-type: none"> ♦ For severe respiratory obstruction

Complications

- Otitis media
- Adenitis
- Meningitis

- Pericarditis
- Pneumonia.

9. Differential diagnosis of postaural abscess.

Differential Diagnosis of Postaural Abscess

	Location	Features
Postauricular (subperiosteal abscess)	♦ Over MacEwen's triangle	♦ Increased intensity of pain over mastoid antrum ♦ Pinna displaced forward, outward and downward (erection of pinna)
Zygomatic abscess	♦ Front of and above pinna, either superficial or deep to temporalis muscle	♦ Associated with edema of upper eyelids
Bezold abscess	♦ Deep to anterior border of sternomastoid, pushing muscle outwards ♦ Between tip of mastoid and angle of jaw by follows posterior belly of digastric ♦ Upper part of posterior triangle ♦ Lower down in neck along carotid vessels	♦ Sudden onset ♦ Pain at back of ear or neck ♦ Fever ♦ Tender brawny swelling in upper part of neck ♦ Torticollis ♦ Abscess may extend downward within sheath of muscle (sinking abscess)
Meatal abscess (Luc's abscess)	♦ Deep part of bony meatus	♦ Pus breaks through bony wall between antrum and external osseous meatus ♦ May burst into meatus
Citelli's abscess	♦ Behind mastoid, more towards occipital bone	

10. Retropharyngeal abscess.

Refer Question No. 2 December 2009 (RS2).

11. Bronchopulmonary segments.

- Bronchopulmonary segments are well-defined sectors of lung, each one of which is aerated by a tertiary or segmental bronchus
- Each segment is pyramidal in shape with its apex directed towards root of lung.

Numbers

- Right lung has ten bronchopulmonary segments and left has eight which are constant in number and position.

Right Lung		Left Lung	
a. Three in upper lobe	i. Apical ii. Anterior iii. Posterior	a. Four in upper lobe Upper division	i. Apicoposterior ii. Anterior
b. Two in middle lobe	i. Medial ii. Lateral	Lower lingular division	i. Superior ii. Inferior
c. Four in lower lobe	i. Superior ii. Anterior basal iii. Posterior basal iv. Lateral basal	b. Four in lower lobe	i. Superior or Apical ii. Anteromedial basal iii. Posterior basal iv. Lateral basal

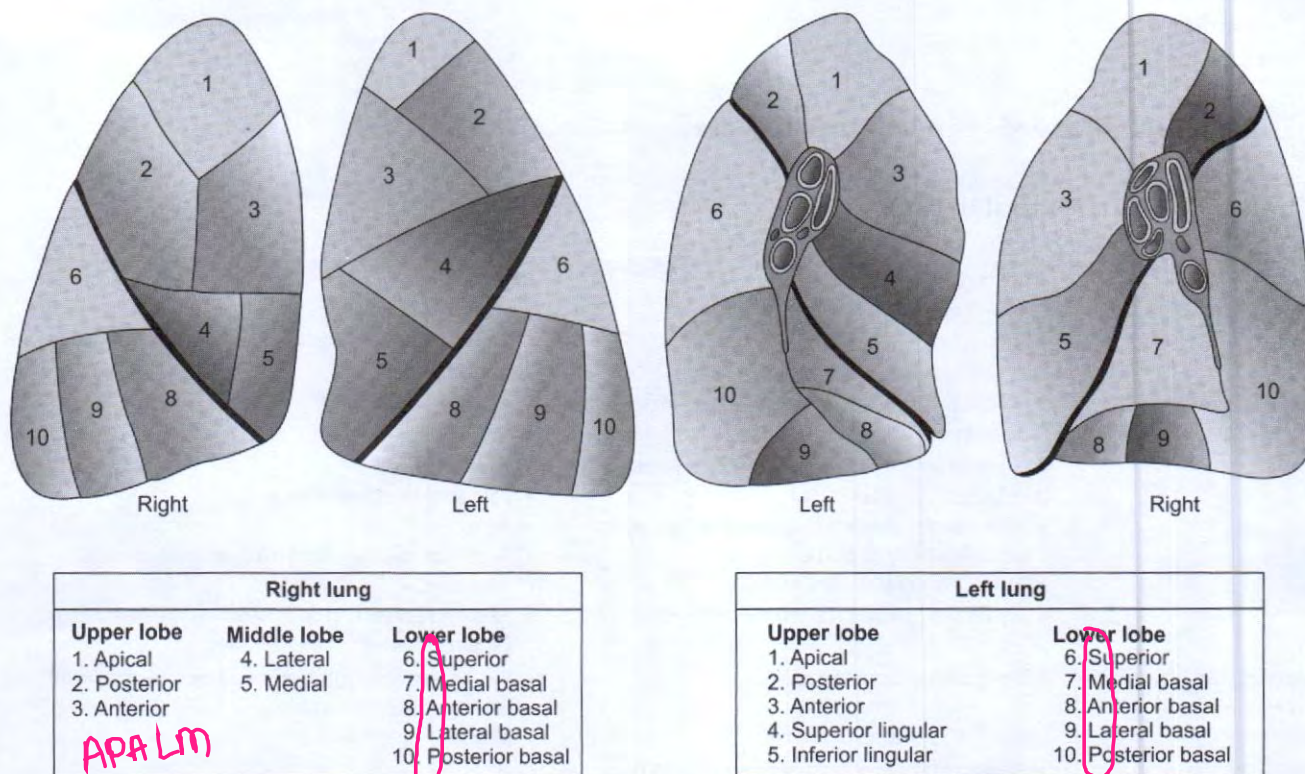


Figure 8: Bronchopulmonary segments

Features

- Bronchopulmonary segments are independent respiratory units
- Each segment is separated from other by intersegmental areolar septa which are occupied by intersegmental tributaries of pulmonary vein
- Pulmonary artery is segmental in distribution and is accompanied with tertiary bronchus
- Thus, each segment has one artery but can have more than one vein.

Applied Anatomy

- Usually, infection of a segment remains restricted to it although tuberculosis may spread from one segment to another
- Bronchiogenic carcinoma breaks through this fibroareolar septa and spread widely
- Knowledge of the detailed anatomy of bronchial tree helps in:
 - Surgical removal of a segment
 - Drainage of infection by adapting different postures (postural drainage)
 - Visualizing the interior of the bronchi by bronchoscope
 - In understanding why abscess are common in some segments.

12. Pharyngocutaneous fistula.

- Pharyngocutaneous fistula is serious and relatively common complication of head and neck surgery.

Etiology

Etiology	Predisposing factors
<ul style="list-style-type: none"> ♦ Postoperative complication of <ul style="list-style-type: none"> – Total laryngectomy – Resection of oropharyngeal carcinoma ♦ Penetrating neck injury 	<ul style="list-style-type: none"> ♦ Poor nutritional status ♦ Prior irradiation ♦ Poor surgical technique ♦ Factors impairing wound healing like diabetes, preoperative hypoalbuminemia, chronic pulmonary diseases and chronic hepatopathy

Clinical features

- Usually seen 1–6 weeks after surgery.

Symptoms	Signs
<ul style="list-style-type: none"> ♦ H/O operation in neck ♦ Pain in lower neck ♦ Associated with fever ♦ Discharge of saliva and purulent material from incision 	<ul style="list-style-type: none"> ♦ Erythema and tenderness in lower neck incision or skin flap (initially) ♦ Sloughing of neck skin exposing neural and vascular structures in neck (after few days)

Investigations

Methylene blue swallowing test	Blood investigations
<ul style="list-style-type: none"> ♦ Confirms communication from skin to pharynx 	<ul style="list-style-type: none"> ♦ Leukocytosis

Prevention

- Gentle atraumatic handling of the soft tissues
- Achieving a watertight anastomosis
- Ensuring complete hemostasis
- Using closed suction drains to eliminate dead space.

Treatment

	Conservative	Operative
Indications	<ul style="list-style-type: none"> ♦ Small fistulae 	<ul style="list-style-type: none"> ♦ Massive fistulae ♦ Chronic, nonhealing fistulae
Techniques	<ul style="list-style-type: none"> i. Local wound care (antiseptic dressing, minimal debridement and antibiotics) ii. Parenteral feeding or nasogastric or gastrostomy tube feeding, with patient nil by mouth 	<ul style="list-style-type: none"> i. Debridement and local closure ii. Regional tissue transfer (mucocutaneous pedicle flap) iii. Free tissue transfer

■ SHORT ANSWERS**13. Foreign bodies in the ear.**

- Foreign bodies in ear may be living or nonliving.

Etiology (Classification)

Animate	Inanimate	
	Organic	Inorganic
<ul style="list-style-type: none"> ♦ Maggots 	<ul style="list-style-type: none"> ♦ Seeds ♦ Wood ♦ Paper 	<ul style="list-style-type: none"> ♦ Stone ♦ Bead ♦ Plastic ♦ Metal

Clinical Features

- Common in children or schizophrenics.

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Pain ♦ Irritation ♦ Crawling sensation (living foreign body) ♦ Noise in ear (living foreign body) ♦ Deafness (vegetable foreign body) 	<ul style="list-style-type: none"> ♦ Foreign body visible in meatus ♦ Inability to see tympanic membrane completely

Treatment

<i>Animate</i>	<i>Conservative</i>		<i>Operative</i>
	<i>Inanimate – organic</i>	<i>Inanimate – inorganic</i>	
Oil/chloroform/ethyl chloride instillation to immobilization of maggots followed by removal using forceps or hook	Absolute alcohol/glycerine instillation to shrink hygroscopic foreign body followed by removal using hook or forceps	<ul style="list-style-type: none"> ♦ Removal using hook or forceps ♦ Syringing 	Removal using postaural/endaural approach <i>Indications</i> <ul style="list-style-type: none"> ♦ Impacted foreign bodies ♦ Inadequate exposure (medial to isthmus) ♦ Inadequate space for manipulation ♦ Foreign body pushed into middle ear

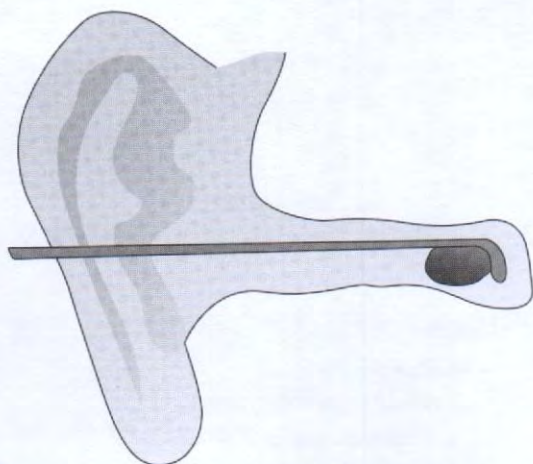


Figure 9A: Removal of foreign body in ear using wax hook

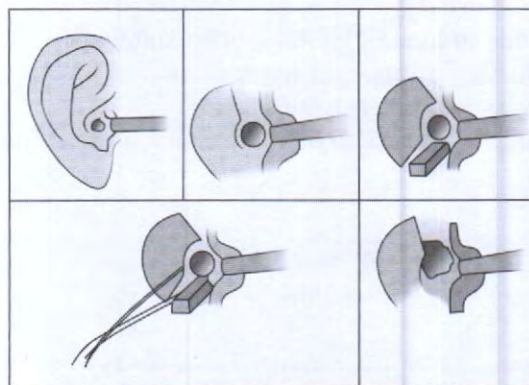


Figure 9B: Endaural approach

Syringing

- A technique of removing foreign body from ear using water.

Principle

- Liquid in a narrow closed cavity built a pressure at closed end and return.

<i>Indications</i>	<i>Contraindications</i>
<ul style="list-style-type: none"> ♦ Foreign body in ear ♦ Wax in ear ♦ Debris in ear ♦ Discharge cleaning 	<ul style="list-style-type: none"> ♦ Perforated tympanic membrane ♦ History of previous ear discharge (indicates tympanic membrane perforation) ♦ CSF otorrhea ♦ History of head injury (may cause fracture line connecting middle ear and intracranial fossa) ♦ Vegetable foreign body (may swell on contact with water) ♦ Living foreign body

Procedure

- Make patient to sit or lie down with ear to be syringed towards doctor, slightly tilted downwards to collect return fluid
- Place a towel around his neck
- Ask patient to hold kidney tray over his shoulder or on bed
- Pull pinna upward and backward and using aural syringe direct a stream of water (normal saline) along posterosuperior wall of meatus
- Foreign body would be expelled by return gush of water into kidney tray.

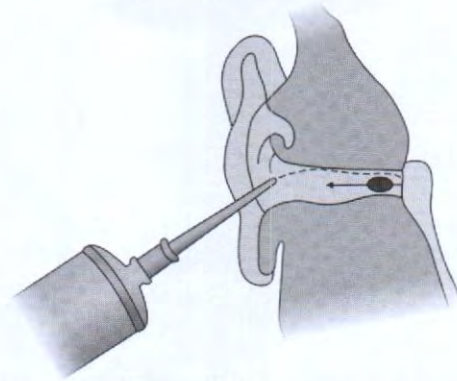


Figure 10: Syringing

Follow-up

- Mop dry ear canal and tympanic membrane with cotton and inspect for any residual foreign body or any trauma
- Instill antibiotic drops into ear canal.

Precautions	Complications
<ul style="list-style-type: none"> ♦ Always use boiled tap water (to sterilize it) cooled to body temperature (because hot water may burn meatal skin and cold water may simulate vagus resulting in vasovagal shock) ♦ Use optimal force while syringing (as excessive force may rupture tympanic membrane) ♦ Create a space between foreign body and meatal wall for jet of water to pass (otherwise syringing may be ineffective or further impacted deeply) 	<ul style="list-style-type: none"> ♦ Traumatic perforation of tympanic membrane (if excessive force is used) ♦ Vasovagal attack (due to stimulation of vagus) ♦ Further impaction of foreign body ♦ Vertigo (due to labyrinthine stimulation) ♦ Infection (if unsterilized water used)

14. Glomus tympanicum.

Refer Question No. 6 December 2013 (RS2).

15. Posterior nasal packing.

Refer Question No. 1 June 2009 (RS2).

16. Complications of sinusitis.

- Spread of infection into or beyond bony wall of sinus is termed complication of sinusitis.

Complications of Sinusitis

Local	Orbital	Intracranial	Others
<ul style="list-style-type: none"> ♦ Mucocele/mucopyocele ♦ Osteomyelitis (frontal bone/maxilla) ♦ Mucous retention cyst 	<ul style="list-style-type: none"> ♦ Orbital cellulitis ♦ Orbital abscess ♦ Orbital apex syndrome ♦ Preseptal inflammatory edema of lids ♦ Subperiosteal abscess ♦ Superior orbital fissure syndrome 	<ul style="list-style-type: none"> ♦ Brain abscess ♦ Extradural abscess ♦ Subdural abscess ♦ Thrombosis of cavernous sinus ♦ Meningitis 	<ul style="list-style-type: none"> ♦ Descending infections ♦ Focal infections
"MOM"	"OOPSS"	"BEST Mnemonics"	

17. Complications of submucosal resection.

Refer Question No. 12 June 2014 (RS2).

18. Sphenoid sinus.

Refer Question No. 2 June 2009 (RS2).

19. Choanal atresia.

Refer Question No. 7 December 2013 (RS2).

20. Posterior epistaxis.

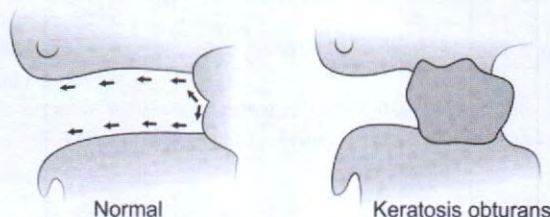
Refer Question No. 1 June 2009 (RS2).

21. Keratosis obturans.

- Keratosis obturans is a collection of pearly white mass of desquamated epithelial cells in deep meatus
- Also called canal wall cholesteatoma.

Etiopathogenesis

<i>Etiology</i>	<i>Predisposing factors</i>	<i>Pathogenesis</i>
♦ Abnormal desquamation of epithelium in deep external auditory canal	♦ Cerumen (accumulation of wax) ♦ Bronchiectasis ♦ Chronic sinusitis	♦ Failure of migration or obstruction to migration of surface epithelium of tympanic membrane to posterior meatal wall → accumulation of epithelial debris → epithelial plug

**Figure 11:** Keratosis obturans—pathogenesis**Clinical Features**

- Common in age between 5 and 20 years
- May be unilateral or bilateral.

<i>Symptoms</i>	<i>Signs</i>
♦ Severe pain ♦ Conductive deafness ♦ Tinnitus ♦ Ear discharge (sometimes)	♦ Pearly white mass of keratin material disposed in several layers ♦ Widening of bony meatus with ulceration and granulation formation

Treatment***Conservative***

- Removal of keratotic mass
 - Either by syringing or instrumentation.
- 2% salicylic acid in alcohol
 - To prevent recurrence or in recurrent cases.

Complications

- Secondary otitis externa
- Facial nerve paralysis (due to widening of meatus because of bony erosion by pressure effect).

22. Vocal cord polyps.

- Vocal polyps are most common benign lesions of larynx.

Etiopathogenesis

Etiology	Predisposing factors	Pathogenesis
<ul style="list-style-type: none"> Vocal abuse or misuse 	<ul style="list-style-type: none"> Allergy Smoking Upper respiratory tract infection 	<p>Sudden shouting</p> <p>↓</p> <p>Hemorrhage in vocal cord</p> <p>↓</p> <p>Submucoal edema</p> <p>↓</p> <p>Vocal polyp</p>

Pathology

Gross appearance	Microscopic examination
<ul style="list-style-type: none"> Soft, smooth, pedunculated swelling May be bright reddish purple to pale translucent 	<ul style="list-style-type: none"> Relatively acellular scanty stroma distended with mucoid exudates Fibrosis and fibrinoid and hyaline degeneration in stroma as lesion matures



Figure 12: Vocal polyp

Clinical Features

- Mostly affects males in age group of 30–50 years.

Symptoms	Signs
<ul style="list-style-type: none"> Hoarseness of voice Double voice (diplophonia) due to different vibratory frequencies of two vocal cords Sticky sensation in throat Dyspnea, stridor or intermittent choking (in case of large polyp) 	<ul style="list-style-type: none"> Unilateral soft, smooth, pedunculated or sessile swelling Arises from junction of anterior 1/3rd and posterior 2/3rd of vocal cord May be bright reddish purple to pale translucent May flop up and down glottis during respiration or phonation

Treatment

Conservative	Operative
<ul style="list-style-type: none"> Speech therapy 	<ul style="list-style-type: none"> Excision by microlaryngosurgery

MBBS PHASE III EXAMINATION

DECEMBER 2008

(Revised Scheme 2)

LONG ESSAYS

1. Discuss the physiological basis and mechanism of deglutition.

- Deglutition or swallowing is a process by which chewed food is emptied from mouth into stomach
- It is initiated voluntarily but ends reflexly.

Stages

Oral stage	Pharyngeal stage	Esophageal stage
Voluntary stage pushing food from mouth into pharynx	Involuntary stage pushing food from pharynx into esophagus	Involuntary stage transporting food into stomach through series of peristaltic waves in esophagus

- a. Oral stage or first stage
- Voluntary stage where food enters from mouth into pharynx.

Mechanism

- Once bolus of food is formed, it is projected on to back of the tongue
 - Tongue is elevated and pressed against hard palate and moved backwards
 - Soft palate is elevated and bolus is propelled into pharynx.
- b. Pharyngeal stage or second stage
- Involuntary stage pushing bolus of food from pharynx into esophagus
 - Also called swallowing reflex
 - Receptors of this reflex are present in vicinity of anterior and posterior pillars of fauces and tonsils
 - Impulse is sent to swallowing center in medullary reticular formation via trigeminal and glossopharyngeal nerves
 - Vth, VIth, IX, X and XII nerves carry motor impulse from swallowing center
 - This reflex also inhibits respiration, sneezing, coughing and vomiting during this stage.

Mechanism

- Once bolus enters pharynx, it can enter 4 ways which are:

♦ Back into mouth	♦ Upward into nasopharynx	♦ Forward into larynx	♦ Downward into esophagus
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- Various movements are coordinated, so that bolus enters only into esophagus
- Entry of bolus in other ways is prevented as follows:

Back into mouth prevented by	Upward into nasopharynx prevented by	Forward into larynx prevented by
<ul style="list-style-type: none">♦ Position of tongue against roof of mouth♦ High intraoral pressure developed by movements of tongue	<ul style="list-style-type: none">♦ Elevation of soft palate	<ul style="list-style-type: none">♦ Approximation of vocal cords♦ Forward and upward movements of larynx♦ Backward movement of epiglottis to close larynx♦ Deglutition apnea which is temporary unrest of breathing occurring in this stage

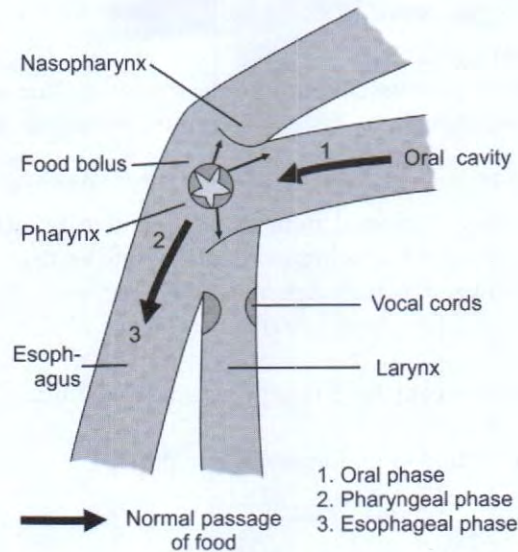


Figure 1: Phase of deglutition

- Entry of bolus into the esophagus is favored by:
 - Closure of rest 3 outlets
 - Stretched opening of larynx by upward movement of larynx
 - Relaxation of the upper esophageal sphincter
 - Peristaltic movements of pharynx
 - Elevation of the larynx lifts the glottis away from the food passage
- Above all movements occur simultaneously in a coordinated fashion to facilitate entry of bolus into esophagus in this 2nd stage of deglutition which lasts only for 1-2 seconds.
- c. Esophageal stage or third stage
 - Involuntary stage where bolus of food is transported to stomach by a series of peristaltic waves in esophagus.

Mechanism

- Two types of peristaltic waves occurring in esophagus are primary and secondary peristaltic contractions.

Primary peristaltic contractions	Secondary peristaltic contractions
<ul style="list-style-type: none"> ◆ These start when bolus reaches upper part of esophagus ◆ These contractions pass downward and propel bolus towards stomach by developing a pressure 	<ul style="list-style-type: none"> ◆ Arise locally in esophagus due to distention of upper esophagus by bolus ◆ Travel downward producing a positive pressure (similar to primary peristalsis) ◆ Waves appear even in absence of primary peristaltic waves and push bolus into stomach

Significance

- Deglutition is first and an important process in gastrointestinal system helping food to move from mouth into stomach.

2. Discuss the etiopathology, clinical features and management of Meniere's disease.

- Meniere's disease is a disorder of inner ear characterized by vertigo, sensorineural hearing loss, tinnitus and aural fullness
- Also called as endolymphatic hydrops, as it causes distension of endolymphatic system
- Named after French physician Meniere who first described it in 1861.

Etiopathogenesis

- a. Defective absorption by endolymphatic sac
 - Ischemia of endolymphatic sac → poor vascularity → defective absorption of endolymph → increased volume of endolymph → distension of endolymphatic system → rupture of Reissner's membrane → mixing of perilymph and endolymph → vertigo.
- b. Vasomotor disturbances
 - Sympathetic overactivity → spasm of internal auditory artery and its branches → interference with functioning of cochlear or vestibular sensory neuroepithelium → deafness and vertigo
 - Anoxia of stria vascularis capillaries → increased permeability → increased transudation → increased production of endolymph → distension of endolymphatic system.
- c. Allergy
 - Allergen (food or inhalant) → inner ear (shock organ) → increased production of endolymph.
- d. Sodium and water retention
 - Retention of excessive amount of fluid → endolymphatic hydrops.
- e. Hypothyroidism.
- f. Autoimmune and viral etiologies.

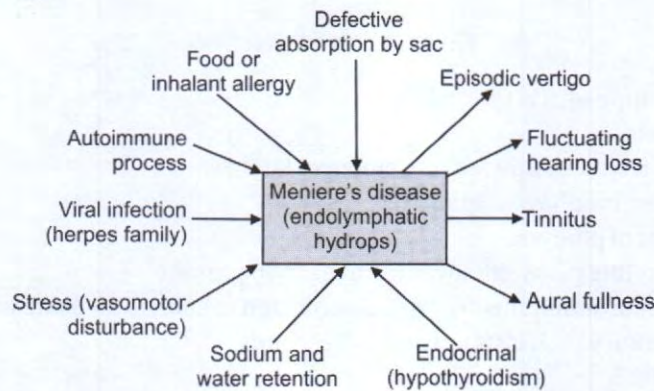


Figure 2: Meniere's disease—etiology and clinical features

Pathology

- Distension of endolymphatic system mainly affecting cochlear duct and saccule and to a lesser extent utricle and semicircular canals
- Complete filling of scala vestibule → marked bulging of Reissner's membrane → herniation of Reissner's membrane through helicotrema into apical part of scala tympani
- Distended saccule lie against stapes footplate
- Utricle and saccule show outpouching into semicircular canals.

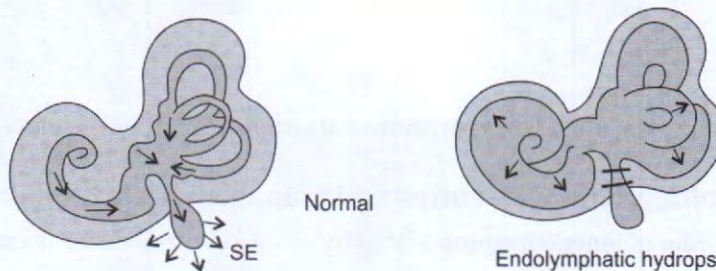


Figure 3: Meniere's disease—pathology

Clinical Features

- Commonly affects males in 35–60 years age group
- Unilateral (50%), but affects other ear in few years.

Symptoms

a. Giddiness (Vertigo)	<ul style="list-style-type: none"> ◆ Episodic attacks in clusters with periods of spontaneous remissions lasting for weeks, months or years ◆ Sudden in onset of varying intensity, duration and interval ◆ Feeling of rotation or “to and fro” or “up and down” movements ◆ Accompanied by: <ul style="list-style-type: none"> – Nausea, vomiting, ataxia and nystagmus (mild attacks) – Abdominal cramps, diarrhea, cold sweats, pallor and bradycardia (severe attacks) ◆ Sometimes, vertigo preceded by sense of fullness in ear, change in character of tinnitus or discomfort in ear ◆ Sometimes, loud sounds or noise induce vertigo (Tullio phenomenon) due to distended saccule lying against footplate of stapes
b. Sensorineural hearing loss	<ul style="list-style-type: none"> ◆ Fluctuating ◆ Accompanies or precedes vertigo ◆ Improves after attack and normal during remissions ◆ With recurrent attacks, incomplete improvement after attacks leading to slow, progressive and permanent deterioration of hearing ◆ Distortion of sound (tone of sound heard normally in one ear but with higher pitch in other ear) ◆ Intolerance to loud sound (due to recruitment phenomenon)
c. Tinnitus	<ul style="list-style-type: none"> ◆ Low pitched roaring type (sometimes hissing character) ◆ Aggravated during acute attacks ◆ Persists during periods of remission ◆ Acute attack may be preceded by change in intensity and pitch of tinnitus
d. Sense of fullness or pressure	<ul style="list-style-type: none"> ◆ Accompanies or precedes an attack
e. Other features	<ul style="list-style-type: none"> ◆ Emotionally upset due to apprehension of repetitive attacks

Signs

Nystagmus	During acute attack Quick component towards unaffected ear
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Investigations

a. Tuning fork tests (reveals sensorineural hearing loss)	i. Rinnes test – Positive (AC > BC) ii. Weber test – Lateralized to better ear iii. Absolute bone conduction test – Reduced in affected ear
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b. Pure tone audiometry

- ♦ Rising curve at early stage when lower frequencies are affected
- ♦ Flat or falling curve with involvement of higher frequencies

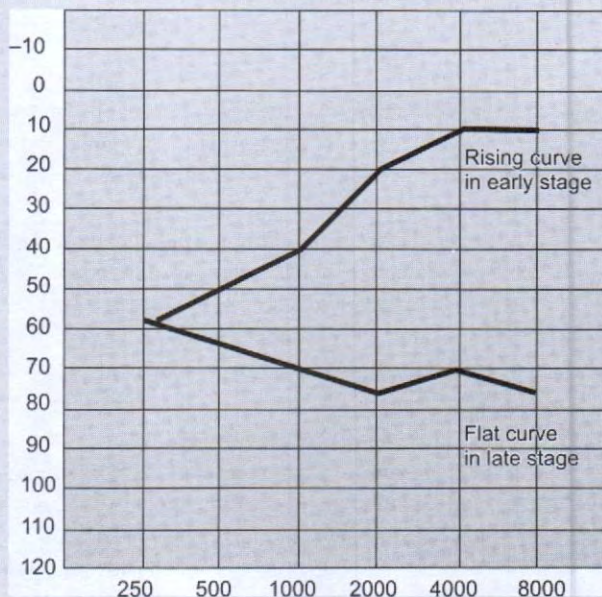


Figure 4: Meniere's disease—audiogram

c. Speech audiometry

- ♦ Discrimination score of 55–85% in remission phase but greatly impaired during and immediately after attack

d. Special audiometry tests (to indicate cochlear lesion and differentiate from retrocochlear lesions)

- Recruitment test
 - Positive
- Short increment sensitivity index (SISI) test
 - >70%
- Threshold tone decay test
 - <25 dB

e. Electrocochleography

- ♦ Ratio of summing potential to action potential >30% (normal: 30%)

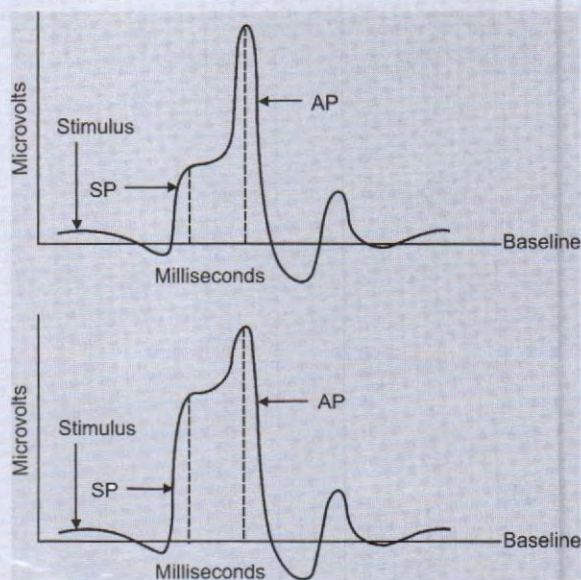


Figure 5: Meniere's disease—electrocochleograph

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f. Calorie test	<ul style="list-style-type: none"> Reduced response on affected side (75%) Reveals canal paresis on affected side
g. Glycerol test	<p>Principle</p> <ul style="list-style-type: none"> Glycerol is a dehydrating agent which reduces endolymph pressure and causes improvement in hearing <p>Procedure</p> <ul style="list-style-type: none"> Record audiogram and speech discrimination score Give 1.5 mL/kg of 95% glycerol with equal amount of water and little flavouring agent Record audiogram and speech discrimination score at hourly interval for 2–3 hours <p>Interpretation</p> <ul style="list-style-type: none"> Positive results means improvement of 10 dB in two or more adjacent octaves or gain of 10% discrimination score There is also improvement in tinnitus and sense of fullness in ear <p>Significance</p> <ul style="list-style-type: none"> Test has diagnostic and prognostic value

Differential Diagnosis

<i>Vestibular neuritis</i>	<i>Benign paroxysmal positional vertigo</i>	<i>Acoustic neuroma</i>
<ul style="list-style-type: none"> Acute onset vertigo Recurrent vertigo not usual No sensorineural deafness No tinnitus No recruitment phenomenon 	<ul style="list-style-type: none"> Recurrent, momentary, positional vertigo Not associated with nausea and vomiting No sensorineural deafness No tinnitus No recruitment phenomenon 	<ul style="list-style-type: none"> Chronic unsteadiness Vertigo, neither marked nor paroxysmal Progressive unilateral sensorineural deafness No recruitment Associated cranial nerve palsies

Treatment

Conservative

A. Acute attack

<i>Supportive</i>	<i>Specific</i>
<ul style="list-style-type: none"> Reassurance and psychological support to allay worry and anxiety Bed rest with head supported on pillow to avoid excessive movements 	<p>a. Vestibular sedatives (to relieve vertigo)</p> <ul style="list-style-type: none"> 15–75 mg prochlorperazine daily orally or IM 25 mg chlorpromazine TID 5–10 mg diazepam IV (tranquillizing effect and suppresses activity of medial vestibular nucleus) 0.4 mg atropine SC <p>b. Vasodilators</p> <p>i. Carbogen inhalation (5% CO₂ and 95% O₂)</p> <ul style="list-style-type: none"> Good cerebral vasodilator and improves labyrinthine circulation <p>ii. Histamine drip</p> <ul style="list-style-type: none"> 2.75 mg histamine diphosphate dissolved in 500 mL glucose by IV drip

B. Chronic phase

<i>Supportive</i>	<i>Specific</i>
<ul style="list-style-type: none"> Reassurance, particularly important during acute attack to anxious patient Cessation of smoking to prevent vasospasm by nicotine Low salt diet (limit salt intake to 1.5–2 g/day) Avoid excessive consumption of water Avoid tea, coffee and alcohol 	<p>a. Vestibular sedatives</p> <ul style="list-style-type: none"> 10 mg prochlorperazine TID for 2 months, then reduced to 5 mg TID for 1 month <p>b. Vasodilators</p> <ul style="list-style-type: none"> 50 mg nicotinic acid TID just before meals 8–16 mg betahistine TID <p>c. Diuretics</p> <ul style="list-style-type: none"> 40 mg furosemide on alternate day along with potassium supplement (if not controlled by vestibular sedatives or vasodilators)

Contd...

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- ♦ Avoid stress and prefer relaxation exercises like yoga, meditation
- ♦ Avoid activities requiring good body balance like flying, etc.
- d. Propantheline bromide
 - 15 mg TID alone or in combination with vasodilator
- e. Elimination of allergen
- f. Hormone replacement
- g. Chemical labyrinthectomy
 - Intratympanic injection of gentamicin daily or biweekly into middle ear to cause destruction of vestibular labyrinth

Operative**Indications**

- Failure of conservative treatment.

Conservative procedures	Destructive procedures
Indications <ul style="list-style-type: none"> ♦ Hearing still useful with disabling vertigo Techniques <ol style="list-style-type: none"> a. Decompression of endolymphatic sac b. Endolymphatic shunt operation <ul style="list-style-type: none"> – Involves insertion of a tube connecting endolymphatic sac with subarachnoid space to drain excess endolymph c. Sacculotomy (Fick's operation) <ul style="list-style-type: none"> – Involves puncturing sacculle with a needle through footplate of stapes d. Cody's tack procedure <ul style="list-style-type: none"> – Involves placement of a stainless steel tack through footplate of stapes to cause periodic decompression of sacculle when it gets distended e. Cochleosacculotomy (otic-periotic shunt) <ul style="list-style-type: none"> – Involves puncturing cochlear duct by a curve needle passed through round window to drain it into perilymph f. Section of vestibular nerve <ul style="list-style-type: none"> – Selective sectioning of vestibular nerve by exposing it through retrosigmoid or middle cranial fossa approach g. Ultrasonic destruction of vestibular labyrinth h. Cervical sympathectomy (to correct microcirculatory fault in labyrinth) 	Indications <ul style="list-style-type: none"> ♦ Unserviceable cochlear functions Techniques <ul style="list-style-type: none"> ♦ Labyrinthectomy <ul style="list-style-type: none"> – Involves complete destruction of membranous labyrinth by opening through lateral semicircular canal by transmastoid route or through oval window by transcanal approach

Recent Advances

- Intermittent low-pressure pulse therapy (Meniett device therapy)
 - Delivery of intermittent positive-pressure waves produced by an instrument called Meniett device (placed in external ear) to round window membrane through a ventilation tube inserted after myringotomy
 - Pressure waves produced pass through perilymph and reduce endolymph pressure by redistributing it through various communication channels.

Advantages

- Nondestructive
- Self administrable.

SHORT ESSAYS**3. Conductive deafness.**

- Conductive deafness or hearing loss is due to defect in sound conducting mechanism anywhere between external auditory canal and footplate of stapes.

Causes

<i>Congenital</i>	<i>Acquired</i>				
	<i>External ear</i>	<i>Tympanic membrane</i>	<i>Middle ear</i>	<i>Eustachian tube</i>	<i>Systemic causes</i>
<ul style="list-style-type: none"> ♦ Meatal atresia ♦ Fixation of footplate of stapes/ head of malleus ♦ Ossicular discontinuity ♦ Congenital cholesteatoma ♦ Congenital absence of oval window 	<ul style="list-style-type: none"> ♦ Wax (MC) ♦ Otitomycosis ♦ Otitis externa ♦ Foreign bodies ♦ Polyps ♦ Traumatic stenosis ♦ Keratosis obturans ♦ Tumors (osteoma, exostosis) 	<ul style="list-style-type: none"> ♦ Bullous myringitis ♦ Traumatic rupture ♦ Perforation due to middle ear infection 	<ul style="list-style-type: none"> a. Traumatic <ul style="list-style-type: none"> – Barotrauma – Hemotympanum – Ossicular discontinuity – Fracture base of skull b. Inflammatory <ul style="list-style-type: none"> – ASOM – CSOM – Serous OM – Adhesive OM c. Chronic infections <ul style="list-style-type: none"> – Tuberculous OM – Syphilitic OM d. Neoplasms (rare) <ul style="list-style-type: none"> – Glomus jugulare – Squamous cell carcinoma e. Miscellaneous <ul style="list-style-type: none"> – Otosclerosis 	<ul style="list-style-type: none"> ♦ Eustachian catarrh (Very common) ♦ Eustachian tube dysfunction (due to enlarged adenoids, nasopharyngeal growth, etc.) ♦ Barotrauma 	<ul style="list-style-type: none"> ♦ Wegener's granulomatosis ♦ Relapsing polychondritis ♦ Fibrous dysplasia ♦ Eosinophilic granuloma ♦ Sarcoidosis

Clinical Features

<i>Symptoms</i>	<i>Signs</i>
<ul style="list-style-type: none"> ♦ Deafness ♦ Aural fullness ♦ Pain ♦ Associated with tinnitus, vertigo 	<ul style="list-style-type: none"> ♦ Otoscopy may reveal pathology in external ear or tympanic membrane ♦ Bone conduction better than air conduction ♦ Good speech discrimination

Degree of Deafness (Social Classification)

♦ Normal hearing	0–20 dB
♦ Mild deafness	20–40 dB
♦ Moderate deafness	40–60 dB
♦ Severe deafness	60–80 dB
♦ Profound deafness	>80 dB

Investigations

<i>Tuning fork tests</i>			<i>Audiometry</i>	<i>Radiography</i>
Rinne's test	Weber's test	Absolute bone conduction test		
Negative (BC > AC)	Lateralized to poorer ear	Normal	BC > AC with air-bone gap Loss not more than 60 dB	Schuller's view (X-ray)

Treatment

	<i>Indications</i>	<i>Techniques</i>
Conservative		
Hearing aid	<ul style="list-style-type: none"> ♦ Unfit for surgery ♦ Refusing surgery ♦ Failure of surgery 	

Contd...

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	Indications	Techniques
Operative (principles)		
a. Removal of canal obstruction	<ul style="list-style-type: none"> ◆ Impacted wax ◆ Foreign body ◆ Osteoma or exostosis ◆ Keratosis obturans ◆ Meatal atresia 	
b. Removal of fluid	<ul style="list-style-type: none"> ◆ Acute otitis media ◆ Serous otitis media ◆ Hemotympanum 	i. Myringotomy with or without grommet insertion
c. Removal of mass from middle ear	<ul style="list-style-type: none"> ◆ Middle ear tumors ◆ Cholesteatoma behind intact TM 	i. Tympanotomy followed by removal of mass
d. Repairing TM perforation	<ul style="list-style-type: none"> ◆ Traumatic rupture of TM ◆ Pathological rupture of TM 	i. Myringoplasty ii. Tympanoplasty
e. Restoring ossicular continuity	<ul style="list-style-type: none"> ◆ Otosclerotic fixation of footplate of stapes or head of malleus ◆ Traumatic disruption of ossicular continuity 	i. Stapedectomy ii. Tympanoplasty iii. Ossicular reconstruction

4. Extratemporal complications of chronic suppurative otitis media.

- Extension of infection middle ear (suppurative otitis media) to adjacent structures is common if not properly treated.

Extratemporal (Cervical) Complications

- Subperiosteal abscess
- Zygomatic abscess
- Bezold's abscess
- Luc's abscess
- Citelli's abscess
- Parapharyngeal or retropharyngeal abscess
- Thrombophlebitis of jugular or subclavian vein.

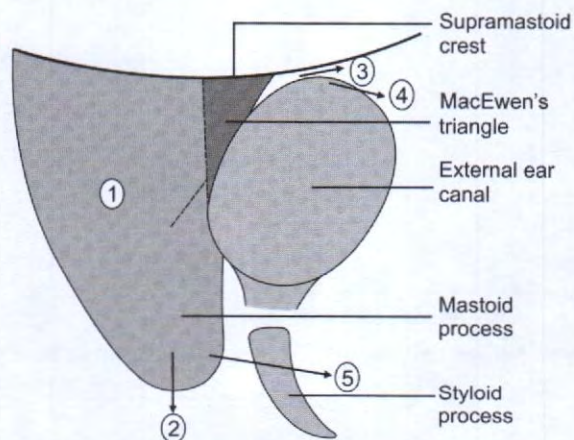


Figure 6: Showing directions of mastoid abscesses (1) Subperiosteal, (2) Bezold's, (3) Zygomatic, (4) Luc's and (5) Citelli's

- a. Postauricular abscess (subperiosteal abscess)
- Commonest abscess forming over mastoid

Location	Clinical features
<ul style="list-style-type: none"> ◆ Over MacEwen's triangle 	<ul style="list-style-type: none"> ◆ Increased intensity of pain over mastoid antrum ◆ Pinna displaced forward, outward and downward (erection of pinna)

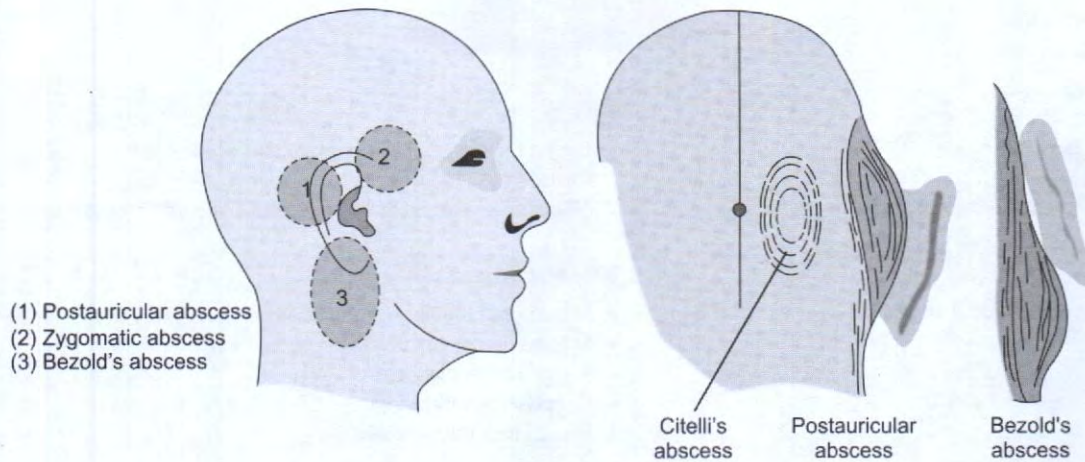


Figure 7: Extratemporal complications

b. Zygomatic abscess

Etiology	Location	Features
♦ Infection of zygomatic air cells situated at posterior root of zygoma	♦ Front of and above pinna, either superficial or deep to temporalis muscle	♦ Associated with edema of upper eyelids

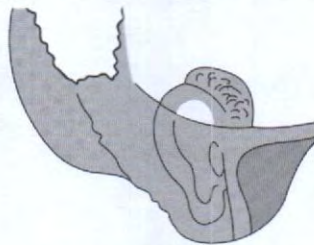


Figure 8: Zygomatic subperiosteal abscess

c. Bezold's abscess

- Bezold's abscess is a subperiosteal abscess formed as a complication of acute coalescent mastoiditis.

Etiology	Location
♦ Perforation and necrosis of medial side of tip of mastoid	<ul style="list-style-type: none"> ♦ Deep to anterior border of sternomastoid, pushing muscle outwards ♦ Between tip of mastoid and angle of jaw by followed posterior belly of digastric ♦ Upper part of posterior triangle ♦ Lower down in neck along carotid vessels

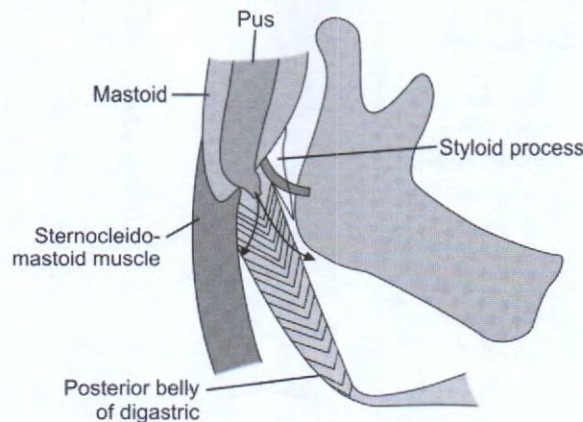


Figure 9: Bezold's abscess

Clinical features

- Sudden onset.

<i>Symptoms</i>	<i>Signs</i>
<ul style="list-style-type: none"> ♦ Pain at back of ear or neck ♦ Fever ♦ H/O purulent ear discharge 	<ul style="list-style-type: none"> ♦ Tender brawny swelling in upper part of neck ♦ Torticollis ♦ Abscess may extend downward within sheath of muscle (sinking abscess)
<i>Investigations</i>	<i>Differential diagnosis</i>
<ul style="list-style-type: none"> ♦ CT scan of mastoid and swelling 	<ul style="list-style-type: none"> ♦ Acute upper jugular lymphadenitis ♦ Abscess or mass in lower part of parotid gland ♦ Infected branchial cyst ♦ Parapharyngeal abscess ♦ Jugular vein thrombosis

*Treatment**Operative*

- Incision and drainage
 - Neck abscess is incised at dependent part and drained using drain
- Treatment of underlying cause
 - Cortical mastoidectomy for coalescent mastoiditis with careful exploration for fistulous opening
 - Intravenous antibiotics based on culture sensitivity at time of I and D.

d. Meatal abscess (Luc's abscess)

<i>Location</i>	<i>Features</i>
<ul style="list-style-type: none"> ♦ Deep part of bony meatus 	<ul style="list-style-type: none"> ♦ Pus breaks through bony wall between antrum and external osseous meatus ♦ May burst into meatus

e. Behind mastoid (Citelli's abscess)

Location

- Behind mastoid, more towards occipital bone.

f. Parapharyngeal or retropharyngeal abscess

- Results from infection of peritubal cells due to acute coalescent mastoiditis.

5. Ototoxicity.

- Ototoxicity is capacity of a drug or chemical to cause functional impairment and cellular degeneration of tissues of inner ear especially end organs and neurons of cochlear and vestibular divisions of VIII nerve.

Etiology (Ototoxic Drugs)

a. Aminoglycoside antibiotics	<ul style="list-style-type: none"> ♦ Cochleotoxic <ul style="list-style-type: none"> – Neomycin – Kanamycin – Framycetin – Tobramycin ♦ Vestibulotoxic <ul style="list-style-type: none"> – Streptomycin – Gentamicin
b. Diuretics	<ul style="list-style-type: none"> ♦ Ethacrynic acid ♦ Furosemide ♦ Bumetanide

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c. Cytotoxic drugs	<ul style="list-style-type: none"> ♦ Cisplatin ♦ Nitrogen mustard ♦ Carboplatin
d. Analgesics	<ul style="list-style-type: none"> ♦ Salicylates ♦ Indomethacin ♦ Phenylbutazone ♦ Ibuprofen
e. Antimalarials	<ul style="list-style-type: none"> ♦ Quinine ♦ Chloroquine
f. Macrolide antibiotics	<ul style="list-style-type: none"> ♦ Erythromycin
g. Glycopeptides antibiotics	<ul style="list-style-type: none"> ♦ Vancomycin
h. Chemicals	<ul style="list-style-type: none"> ♦ Alcohol ♦ Tobacco ♦ Marijuana ♦ Carbon monoxide
i. Miscellaneous	<ul style="list-style-type: none"> ♦ Ampicillin ♦ Propranolol ♦ Propylthiouracil ♦ Deferoxamine ♦ Imipramine ♦ 5-hydroxytryptamine ♦ Carbamazepine

Clinical Features

- Same for all drugs but varies in severity, timing and duration.

Symptoms	Signs
i. Tinnitus <ul style="list-style-type: none"> – First warning symptom – Usually high pitched and continuous – May be unilateral or bilateral ii. Hearing loss <ul style="list-style-type: none"> – Sensorineural deafness affecting high frequencies iii. Vertigo (with vestibulotoxic drugs) <ul style="list-style-type: none"> – Sensation of continuous rotation after turning head or turning over in bed iv. Disequilibrium <ul style="list-style-type: none"> – Severely unsteady and bed ridden in severe cases 	i. Bilateral loss of labyrinthine function (bobbing oscillopsia) <ul style="list-style-type: none"> – On caloric and rotational tests

Investigations

- Estimation of drug concentration in body
- Pure tone audiometry (high tone loss)
- Caloric test
- Electrocochleography
- Otoacoustic emission
- BERA.

Treatment	Prophylaxis
Conservative <ul style="list-style-type: none"> ♦ Mild hypnotics or tinnitus maskers for tinnitus ♦ Reassurance and regular physiotherapy for disequilibrium ♦ Hearing aids or cochlear implants for severe hearing loss 	<ul style="list-style-type: none"> ♦ Early recognition and discontinuation of ototoxic drug ♦ Regular monitoring of serum drug concentration ♦ Avoiding prescription of ototoxic drugs as much as possible ♦ Prescription of drug within maximum recommended doses

6. Nasal Decongestants.

- Nasal decongestants are α -adrenergic agonists producing local vasoconstriction on topical application as dilute solutions

Classification (IP₃)

- Phenylephrine
- Imidazoline compounds
 - Naphazoline
 - Xylometazoline
 - Oxymetazoline
- Pseudoephedrine
- Phenylpropanolamine.

Drug	Adverse effects	Dose
a. Phenylephrine <ul style="list-style-type: none"> A selective α_1 agonist with negligible β action 	<ul style="list-style-type: none"> Reflex bradycardia and rise in BP 	10 mg orally 0.25% topically
b. Imidazoline compounds <ul style="list-style-type: none"> Selective α_2 agonists Longer duration of action 	<ul style="list-style-type: none"> Initial stinging sensation (naphazoline) Impairment of mucociliary function (on prolonged use) Atrophic rhinitis and anosmia (due to persistent vasoconstriction) CNS depressions and rise in BP (on systemic absorption) 	Naphazoline: 0.1% topically Xylometazoline: 0.1% topically Oxymetazoline: 0.05% topically
c. Pseudoephedrine <ul style="list-style-type: none"> Stereoisomer of ephedrine Causes vasoconstriction in mucosa and skin 	<ul style="list-style-type: none"> Few CNS and cardiac effects 	30–60 mg TDS
d. Phenylpropanolamine <ul style="list-style-type: none"> Similar to ephedrine chemically and pharmacologically 	<ul style="list-style-type: none"> Amphetamine like CNS effects 	25–50 mg TDS

Therapeutic Uses

- Used either alone or in combination with antihistaminics, mucolytics, antitussives and analgesics to afford symptomatic relief in common cold, blocked Eustachian tube, upper respiratory tract infection.

7. Role of local anesthesia in otorhinolaryngology.

- Local anesthesia are ideal for short operations like polyp removal or myringotomy, etc. and used often in otorhinolaryngology surgeries.

Ideal local anesthetic	Commonly used local anesthetic
<ul style="list-style-type: none"> Adequate potency Short latency Good penetration Good diffusion Low toxicity Controllable duration of action with complete reversibility Water solubility Soluble in solution Non-irritant, non-antigenic and should not interfere with wound healing 	<ul style="list-style-type: none"> 4% lignocaine with adrenaline

Types

Nerve block	Field block	Local infiltration	Topical analgesia
Consists of depositing a suitable local anesthetic solution within close proximity to a main nerve trunk, thus preventing afferent impulses from traveling centrally beyond that point	Consists of depositing a solution in proximity to the larger terminal nerve branches, so that area to be anesthetized is walled off for circumscribed to prevent central passage of afferent impulses	Consists of flooding of small nerve terminals in area of surgery with local anesthetic solution rendering them insensible to pain or preventing them from becoming stimulated and creating impulse	Consists of application of suitable solution directly to the surface of the area rendering free nerve endings inaccessible structures (intact mucous membrane/abraded skin) incapable of simulation

Techniques

A. Ear

Indications

- Procedures involving the pinna, external auditory canal, tympanic membrane and middle ear

Nerve anesthetized	Procedure
a. Local anesthesia	<ul style="list-style-type: none"> ♦ For myringotomy anesthesia can be obtained by application of 2.5% lignocaine and 2.5% prilocaine (EMLA) cream ♦ For perimeatal operations 0.5 mL of 1% lignocaine just medial to bony and cartilaginous meatus are injected superiorly, inferiorly, posteriorly and anteriorly at same depth and two further injections superiorly and inferiorly 5 mm lateral to the margin of the temporomandibular joint are given ♦ Anesthesia within tympanic cavity can be obtained by placing 4–6 drops of 4% lignocaine
b. Auriculotemporal nerve block	
– Auriculotemporal	♦ Blocked by injecting 1–2 mL lignocaine just anterior to meatus
c. Greater auricular nerve block	
– Greater auricular	<ul style="list-style-type: none"> ♦ Anesthetized behind pinna by raising a wheal in front of the lower anterior border of the mastoid process ♦ A 7 cm needle is inserted and directed upwards and 2 to 3 mL anesthetic solution placed between mastoid process and meatus.

B. Nose and paranasal sinuses

Indications	Procedure
a. Topical Anesthesia	
– Minor surgery to nose, removal of polyps, local electrocautery and antral puncture	<ul style="list-style-type: none"> ♦ Spray nasal cavities with anesthetic ♦ Carefully pack nose with ribbon gauze/cotton soaked in local anesthetic solution for 10 min first at an angle of 20° to floor of nose until bone is felt at a depth of 6–7 cm, now lying adjacent to sphenopalatine foramen and second along anterior border of nasal cavity until anterior end of cribriform plate about 5 cm deep
b. Field Block	
Sites	
– Above nasal bones and beneath skin of dorsum to block infratrochlear nerve	
– Into infraorbital foramen bilaterally to block infraorbital nerve	
– Greater palatine nerve at incisive foramen	
– Nasal tip and base of columella	
c. Anterior ethmoidal nerve block	
– External dacryocystorhinotomy	♦ Anterior ethmoidal nerve and infratrochlear nerves can be blocked together at their origin from nasociliary nerve in the upper half of the medial wall of the orbit 2.5 cm from orbital margin
– Ligation of anterior and posterior ethmoidal arteries for epistaxis (using Lynch incision)	<ul style="list-style-type: none"> ♦ A 5 cm needle with a marker 2.5 cm from tip is inserted 1 cm above inner canthus and directed horizontally backwards ♦ At a depth of 2.5 cm the tip lies close to anterior ethmoidal nerve where it enters its foramen, 1 mL is injected and 1 mL is injected as the needle is slowly withdrawn

C. Tracheobronchial tree

Indications

- To facilitate **diagnostic laryngoscopy**
- **Bronchoscopy**
- Placement of a tracheal tube in patients
- **Blocks of the superior laryngeal nerves bilaterally**, along with translaryngeal injection of local anesthetic, provide anesthesia of the airway from the infraglottic area to the epiglottis, provides satisfactory analgesia for endoscopic procedures.

Nerve anesthetized	Areas anesthetized	Procedure
a. Superior Laryngeal Nerve Block		
– Superior laryngeal nerve	♦ From inferior aspect of epiglottis to vocal cords	<ul style="list-style-type: none"> ♦ Patient is placed supine with neck extended ♦ Hyoid bone is displaced laterally towards side to be blocked and a 25 gauge, 2.5 cm needle is walked off greater cornu of hyoid bone inferiorly and is advanced 2–3 mm ♦ As needle passes through thyrohyoid membrane, a slight loss of resistance is felt and 3 mL of local anesthetic solution is injected superficial and deep to this structure ♦ Block is then repeated on opposite side. This technique produces anesthesia from inferior aspect of epiglottic to vocal cords
b. Translaryngeal Block		
– Recurrent laryngeal nerve	♦ Subglottis and trachea	<ul style="list-style-type: none"> ♦ With patient in supine position, cricothyroid membrane is located and a 20 gauge or smaller, 3–5 cm plastic catheter over a needle is introduced in midline ♦ Inner steel cannula is withdrawn with plastic catheter held firmly in place aspiration of air confirms correct catheter placement ♦ 3–5 mL of 4% lidocaine solution is injected rapidly, usually resulting in a vigorous cough, which aids in spread of solution within trachea
c. Glossopharyngeal Nerve Block		
– Glossopharyngeal nerve	♦ Posterior 1/3rd of tongue, pharynx and superior surface of epiglottis	<ul style="list-style-type: none"> ♦ Can be blocked intraorally by injecting 5 mL of local anesthetic into base of each posterior tonsillar pillar ♦ An angled 22 gauge, 9 cm needle, which can be formed by bending distal 1 cm of a spinal needle with its stylet remove, is employed for this block

D. Head and Neck Surgery

Nerve anesthetized	Areas anesthetized	Procedure
a. Trigeminal nerve block		
<i>Indications</i>		
– Blockade of II and III divisions of the trigeminal nerve is useful in diagnosis and management of pain syndromes and surgical procedures		
b. Maxillary Nerve Block		
– Entire maxillary nerve and all its subdivisions peripheral to site of injection	<ul style="list-style-type: none"> ♦ Anterior temporal and zygomatic regions ♦ Lower eyelid ♦ Side of nose ♦ Anterior cheek ♦ Upper lip ♦ Maxillary teeth ♦ Maxillary alveolar bone and overlying ♦ Hard and soft palate ♦ Tonsil ♦ Part of pharynx ♦ Nasal septum and floor of nose ♦ Posterior lateral mucosa and turbinate bones 	<ul style="list-style-type: none"> ♦ Maxillary nerve is blocked as it exists the skull through foramen rotundum and crosses pterygopalatine or infratemporal fossa between skull and upper jaw ♦ Coronoid notch of mandible is located and with patient's mouth closed, a 22 gauge, 8 cm needle is inserted at inferior edge of coronoid notch perpendicular to skin entry site ♦ Needle contacts lateral pteryoid plate at a depth of about 5 cm ♦ It is then withdrawn and redirected anteriorly and superiorly to walk off plate and is advanced approximately 0.5 cm into pterygopalatine fossa. 3–5 mL of solution is injected

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Nerve anesthetized	Areas anesthetized	Procedure
c. Mandibular Nerve Block		
<i>Indications</i>		
<ul style="list-style-type: none"> Blockade used for diagnosis and therapeutic purpose, such as trigeminal neuralgia Used for surgical procedures where anesthesia of entire distribution of maxillary nerve is required 		
<ul style="list-style-type: none"> Mandibular and its subdivisions Inferior alveolar Buccinator Lingual Mental Incisive 	<ul style="list-style-type: none"> Temporal region Auricle of ear (tragus, root of helix) External auditory meatus TM joint Salivary glands Anterior 2/3rd of tongue Floor of mouth Mandible Lower teeth, gingival and buccal mucosa Lower portion of face 	<ul style="list-style-type: none"> Mandibular nerve leaves cranium through foramen ovale and innervates skin of lower jaw and skin anterior and superior to ear by its posterior division Mandibular nerve is blocked via same entry site as maxillary nerve Needle is advanced along inferior margin of coronoid notch until bone of lateral pterygoid plate is contacted (about 5 cm) Needle is withdrawn and its redirected to walk off posterior border of pterygoid plate, should not be inserted farther than 0.5 cm past plate
<i>Side Effects and Complications</i>		
<ul style="list-style-type: none"> Hematoma formation Temporary blindness Pharynx may be entered, increasing the risk of contaminating the infratemporal fossa Brainstem anesthesia 		
d. Supraorbital and Supratrochlear Nerve Block		
<ul style="list-style-type: none"> Supraorbital and supratrochlear 	<ul style="list-style-type: none"> Whole forehead, upper eyelids 	<ul style="list-style-type: none"> A 25 gauge, 2 cm needle is inserted immediately superior to supraorbital notch and 2–4 mL of local anesthetic solution is injected Supratrochlear nerve can be blocked by extending supraorbital injection site medially with an additional 2–4 mL of solution
e. Infraorbital Nerve Block		
<ul style="list-style-type: none"> Infraorbital Inferior palpebral, lateral nasal and superior labial nerves Anterior and middle superior alveolar nerves 	<ul style="list-style-type: none"> Upper lip, portions of side of nose, lower eyelids Labial alveolar plate and overlying tissues Incisors and bicuspid on side injected Sometimes maxillary molars and their buccal supporting structures 	<ul style="list-style-type: none"> Nerve can be blocked by advancing a 25 gauge, 3 cm needle laterally and cephalic towards foramen from a point 1 cm inferior
f. Mental Nerve Block		
<ul style="list-style-type: none"> Mental 	<ul style="list-style-type: none"> Lower lip Mucous membrane in mucolabial fold anterior to mental foramen 	<ul style="list-style-type: none"> Mental foramen is palpated in mandible and a 25 gauge, 3 cm needle is inserted inferomedially and 1 mL of anesthetic solution is injected
g. Inferior Alveolar Nerve Block		
<ul style="list-style-type: none"> Inferior alveolar and its subdivisions (mental and incisive nerves) 	<ul style="list-style-type: none"> Body of mandible and inferior portion of ramus Mandibular teeth Mucous membrane and underlying tissues anterior to first mandibular molar 	<ul style="list-style-type: none"> Stand to right and front of patient and instruct patient to occlude teeth Retract patient's lips exposing maxillary and mandibular teeth Align syringe parallel to occlusal and sagittal planes, but positioned at mucogingival junction of maxillary molars Penetrate needle in mucosa just medial to ramus and is inserted approximately 1½ inches Aspirate and inject anesthetic

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Nerve anesthetized	Areas anesthetized	Procedure
h. Lingual Nerve Block		
– Lingual	<ul style="list-style-type: none"> ♦ Anterior 2/3 rd of tongue and floor of oral cavity ♦ Mucosa and periosteum on lingual side of mandible 	<ul style="list-style-type: none"> ♦ Stand to right and front of patient and instruct patient to occlude teeth ♦ Retract patient's lips exposing maxillary and mandibular teeth ♦ Align syringe parallel to occlusal and sagittal planes, but positioned at mucogingival junction of maxillary molars ♦ Penetrate needle in mucosa just medial to ramus and is inserted approximately 1½ inches ♦ Aspirate and inject anesthetic
i. Buccinator Nerve Block		
– Buccinator	<ul style="list-style-type: none"> ♦ Buccal mucous membrane and mucoperiosteum of mandibular area 	<ul style="list-style-type: none"> ♦ A 1 inch 25 gauge needle is inserted onto buccal mucosa just distal to third molar and 0.5 mL of solution is deposited ♦ Insert needle and deposit solution directly into retromolar trigone
j. Cervical Plexus Block		
– C1, C2, C3 and C4 spinal nerves	<ul style="list-style-type: none"> ♦ Skin between trigeminally innervated face and T2 dermatome of trunk 	<p>Superficial Cervical Plexus Block</p> <ul style="list-style-type: none"> ♦ Done at midpoint of posterior border of sternocleidomastoid muscle ♦ A skin wheal is made at this point and a 22 gauge, 4 cm needle is advanced, it is an injection of 5 mL of solution along posterior border and medial surface of sternocleidomastoid muscles <p>Deep Cervical Plexus Block</p> <ul style="list-style-type: none"> ♦ Paravertebral block of C2 to C4 spinal nerves as they emerge from their foramina in cervical vertebrae ♦ Usually achieved using three separate injections at C2, C3 and C4 ♦ Patient lies supine with neck slightly extended and head turned away from side to be blocked ♦ A line is drawn connecting tip of mastoid process and Chassaignac tubercle (transverse process of C6); a 2nd line is drawn 1 cm posterior to this first line ♦ After skin wheals are raised over transverse processes of C2, C3 and C4, three 22 gauge, 5 cm needles are advanced perpendicular to skin entry site with a slight caudal angulation ♦ Transverse process is contacted at a depth of 1.5–3 cm, 3–4 mL of solution is injected. ♦ Block can also be performed with a single injection of 10–12 mL, at C4 transverse process ♦ Cephalad spread of local anesthetic usually anesthetizes C2 and C3 nerves
<p>Side Effects and Complications</p> <ul style="list-style-type: none"> – Intravascular injection – Blockade of phrenic and superior laryngeal nerve – Spread of local anesthetic solution into the epidural and subarachnoid space 		

Advantages	Disadvantages (Contraindications)
<ul style="list-style-type: none"> ♦ Patient remains awake and cooperative ♦ Little distortion of normal physiology, thus method can, therefore, be used to advantage on poor risk patients ♦ Functional assessment like hearing gain following ear surgery possible on operation table itself ♦ Low incidence of morbidity ♦ Patient may leave office unescorted ♦ Does not require special skills ♦ Cheap 	<ul style="list-style-type: none"> ♦ Patient refusal ♦ Infection ♦ Allergy to local anesthetics ♦ Incooperative patient ♦ Major and prolonged surgeries ♦ Extremes of age ♦ Anomalies

8. Deaf child.

- Assessment of deaf child is a challenge of otolaryngologist because of detecting deafness before 1 year of age.

Causes

<i>Prenatal</i>	<i>Perinatal</i>	<i>Postnatal</i>
i. Infant factors (inner ear anomalies due to genetic or non-genetic causes)—“ABCDEMS” <ul style="list-style-type: none"> – Alexander’s dysplasia (affliction of basal turn of membranous cochlea affecting only high frequencies) – Bing-Siebert dysplasia (complete absence of membranous labyrinth) – Cochleosaccular (Sheibe’s) dysplasia (dysplasia of cochlea and saccule due to autosomal recessive trait) – Mondini’s dysplasia (presence of only basal coil or 1.5 turns of cochlea) – Enlarged vestibular aqueduct (vestibular aqueduct enlarged >2 mm) – Michel aplasia (complete absence of bony and membranous labyrinth including petrous apex) – Semicircular canal malformations ii. Maternal factors – “DRIO” <ul style="list-style-type: none"> – Drugs (aminoglycosides, thalidomide, chloroquine) – Radiation in 1st trimester – Infections (TORCHES complex) – Others (nutritional deficiency, diabetes, toxemia, alcoholism, thyroid deficiency) 	<ul style="list-style-type: none"> ♦ Birth anoxia ♦ Prematurity and LBW ♦ Birth injuries ♦ Neonatal jaundice ♦ Neonatal meningitis ♦ Sepsis ♦ Ototoxic drugs 	i. Genetic <ul style="list-style-type: none"> – Familial progressive sensorineural deafness – Alport’s syndrome – Hurler’s syndrome ii. Nongenetic <ul style="list-style-type: none"> – Viral infections (measles, mumps, varicella, influenza) – Secretory otitis media – Ototoxic drugs – Trauma (temporal bone fracture, middle ear surgery) – Noise induced hearing loss

Risk factors (Joint Committee on Infant Hearing – 1994)

- Family history
- Prenatal maternal infections (TORCHES complex)
- Craniofacial anomalies including those of pinna and ear canal
- Birth weight <1.5 kg
- Hyperbilirubinemia requiring exchange transfusion
- Ototoxic medications
- Bacterial meningitis
- APGAR score of 0–4 at 1 min and 0–6 at 5 min
- Mechanical ventilation for 5 days or more
- Features of others syndrome associated with deafness.

Clinical Features

<i>Symptoms</i>	<i>Signs</i>
<ul style="list-style-type: none"> ♦ Child sleeps through loud noises unperturbed ♦ Child fails to startle to loud sound ♦ Child fails to develop speech by 1–2 years 	<ul style="list-style-type: none"> ♦ Presence of any of risk factors

Investigations

a. Neonatal screening procedures (tests based on infant’s behavioral response to sound signal to screen high-risk infants)	
i. Arousal test	<ul style="list-style-type: none"> ♦ Presentation of high frequency narrow band noise to infant in light sleep for 2 sec ♦ Normal infants awakens at least twice to three such stimuli
ii. Auditory response cradle	<ul style="list-style-type: none"> ♦ Monitoring of infants behavior (trunk and limb movements, head jerk and respiration) to sound stimuli by transducers in a special cradle
iii. Auditory brainstem response	<ul style="list-style-type: none"> ♦ Assess ability of infant to respond to click stimulus of 40 nHL or less

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b. Behavior observation audiometry (detects change in behavior of infant to sound) (used at birth or child up to 6 months)	
i. Moro's reflex	♦ Sudden movements of limbs and extension of head by infants in response to sound of 80–90 dB
ii. Cochleopalapebral reflex	♦ Responding to loud sound by blinking
iii. Cessation reflex	♦ Stoppage of activity or crying in response to sound of 90 dB
c. Distraction technique (for 6–7 month old children)	
	♦ Detects child's ability to locate source of sound such as high frequency rattle, low frequency hum, etc.
d. Condition technique	
i. Visual reinforcement audiometry (for child aged 5 months to 2 years)	♦ Conditioning of child to look for an auditory stimulus by turning his head to determine hearing threshold using standard audiometric technique
ii. Play audiometry (for child aged 2–5 years)	♦ Conditioning of child to perform an act (placing a marble in box, putting a plastic block in bucket) in response to sound to determine ear specific threshold by standard audiometric technique
iii. Speech audiometry	♦ Asking child to repeat names of certain objects or to point them out on pictures, thus helping to test hearing level and speech discrimination
e. Objective tests	
i. Electrocochleography	♦ An invasive procedure to measure auditory sensitivity within 20 dB
ii. Auditory brainstem response (ABR)	♦ Assess hearing threshold of individual ear of an infant by obtaining ABR tracing at higher sound and gradually lowered till wave V is just identifiable
iii. Otoacoustic emissions	♦ Absence of transient evoked emissions and distortion product emissions indicates hearing loss >30 dB and >50 dB, respectively
iv. Impedance audiometry	♦ Indicates location of pathology

Treatment (Rehabilitation)**Objective**

- Development of speech and language
- Social adjustment
- Vocational training

Specific	Supportive
a. Hearing aids <ul style="list-style-type: none"> – Prescription of hearing aid as early as possible in presence of residual hearing (to amplify them) 	a. Parental guidance <ul style="list-style-type: none"> – Regarding child's disability and its care b. Development of speech and language <ul style="list-style-type: none"> i. Auditory oral communication <ul style="list-style-type: none"> – By using hearing aids, training in speech reading by movements of lips, face, hand and body movements ii. Manual communication <ul style="list-style-type: none"> – By using sign language or finger spelling method iii. Total communication <ul style="list-style-type: none"> – By developing oral speech, lip reading and sign language c. Education <ul style="list-style-type: none"> – Through special schools d. Vocational training <ul style="list-style-type: none"> – Training in appropriate vocation provides a better scope for social adjustment and instills pride among them

- Diagnosis and management of deaf child is very crucial, as there is large improvement in children diagnosed and treated before 6 months of age.

9. Tympanometry.

- Tympanometry is an objective audiometry that measures impedance or resistance offered by conducting mechanism of ear and their compliance to sound pressure transmission
- Also called impedance audiometry.

Principle

- When a sound strikes tympanic membrane, some of sound energy is absorbed while rest is reflected
- Reflection of sound energy by a stiff tympano-ossicular system is more than a compliant one which is assessed by measuring reflected sound energy in a sealed external auditory canal by varying pressure in it.

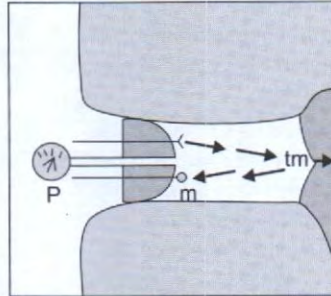


Figure 10: Tympanometry—principle

Components

- A probe with three channels that fit snugly into external auditory canal
 - First probe (oscillator) delivers a tone of 220 Hz
 - Second probe (microphone) picks up reflected sound
 - Third probe (manometer) alters air pressure in meatus.

Procedure

- After fitting probe into external auditory canal, a tone of 220 Hz is delivered and reflected sound energy is recorded under positive (+ 200 mm of H₂O) then normal and finally negative (– 200 mm of H₂O) air pressures with serial reduction of 50 and recording compliance at each change
- Recordings are noted on a graph called tympanogram.

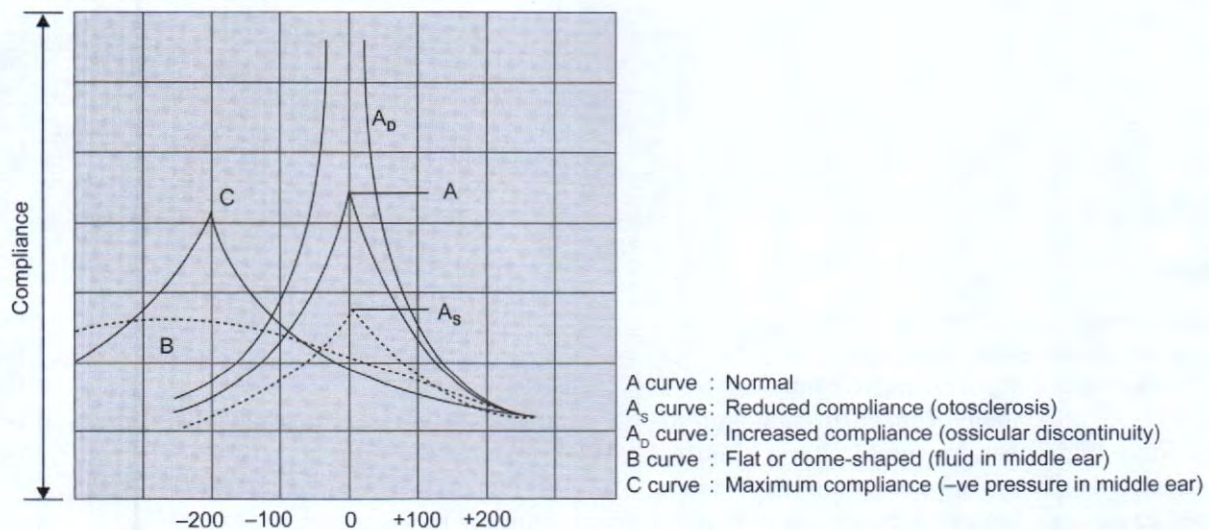
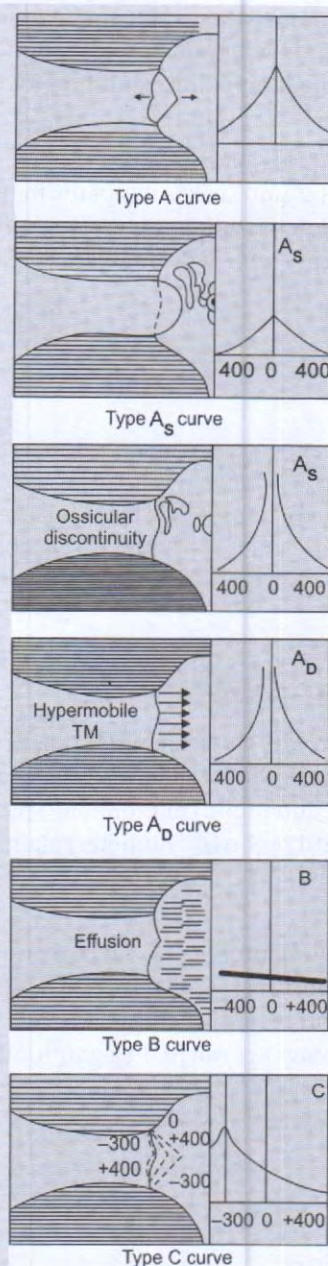


Figure 11: Tympanogram—types

Interpretation

Type of tympanogram	Observation	Diagnosis
a. Type A	More compliance (peak) at normal pressure and decreased compliance at extremes of pressure	Normal tympanogram
b. Type A _s	Lower compliance at or near ambient air pressure	Fixation of ossicles as in otosclerosis, malleus fixation, tympanosclerosis, thick graft in myringoplasty
c. Type A _o	High compliance at or near ambient air pressure	Ossicular discontinuity or thin and lax tympanic membrane or monomeric tympanic membrane
d. Type B	No change in compliance with pressure changes (flat or dome shaped graph)	Impacted wax, foreign body, secretory otitis media, adhesive otitis media, thick tympanic membrane, perforated tympanic membrane, patent grommet
e. Type C	Maximum compliance with pressure > -100 mm of H ₂ O (sharp pointed curve or humped curve)	Retracted tympanic membrane or Eustachian tube obstruction or early secretory otitis media
f. Type D (Liden et al.)	Notched graph	Scarred tympanic membrane, flaccid tympanic membrane

**Figure 12:** Types of tympanogram**Applications**

- Testing compliance of tympanic membrane
- Diagnosis of middle ear pathology
- Assessing functioning of Eustachian tube
 - Done by noting ability of patient to equilibrate middle ear pressure when asked to swallow five times in 20 seconds
 - extreme middle ear pressures (- 200 or + 200 mm of H₂O)
 - Indicated by compliance >2.5 cc in type B curve
- Finding patency of tympanic membrane grommet in serous otitis media
- Eliciting acoustic reflex.

Principle

- Loud sound (70–100 dB above threshold of hearing) causes bilateral contraction of stapedial muscles which can be detected by tympanometry.

Procedure

- Loud tone is delivered to one ear and reflex is picked from same or contralateral ear.

Observation

Findings	Interpretation
♦ Stapedial reflex positive at lower intensities (40–60 dB)	Cochlear type of hearing loss (recruitment)
♦ Stapedial reflex decay (Reflex amplitude reduced to 50% on sustained delivery of 500–1000 Hz tone at 10 dB above acoustic reflex threshold)	VIII nerve lesion (abnormal adaptation)
♦ Absence of stapedial reflex with normal hearing	VIII nerve lesion proximal to nerve to stapedius
♦ Presence of ipsilateral reflex but absence of contralateral reflex	Lesion of crossed pathway in brainstem

Advantages

- Objective test
- Noninvasive
- Useful in children and uncooperative patients
- Assess middle ear function accurately
- Detects malingering
- Differentiates different types of conductive and sensorineural deafness
- Also helps determine level of facial nerve lesion (with reference to nerve to stapedius muscle).

10. Intratemporal course of facial nerve.

- Facial nerve is seventh cranial nerve and nerve of 2nd branchial arch
- Intrapetrous part is part of facial lying within petrous part of temporal bone before it emerges from stylomastoid foramen.

Course and Relations

- Nerve arises as roots, motor and sensory (nervus intermedius), attached to lateral part of lower border of pons just medial to VIII cranial nerve
- Both roots accompany vestibulocochlear nerve and pass laterally and forward to enter internal acoustic meatus where they are accompanied by labyrinthine artery
- In meatus, motor root lodges in a groove on anterosuperior surface of vestibulocochlear nerve with sensory roots intervening in between them
- At bottom of meatus, both roots unite to form a trunk and enters bony facial canal
- In canal, nerve passes laterally above vestibule of internal ear and reaches medial wall of epitympanic part of tympanic cavity where it bends backwards forming a genu
- Here it presents an asymmetrical swelling called genicular ganglion containing cell bodies of pseudo-unipolar sensory neurons
- From genu, nerve passes backward and downward lodging in bony canal above promontory and fenestra vestibuli to reach medial wall of aditus to mastoid antrum to lie below bulging of lateral semicircular canal
- Finally, nerve passes vertically downward along posterior wall of tympanic cavity and leaves temporal bone through stylomastoid foramen.

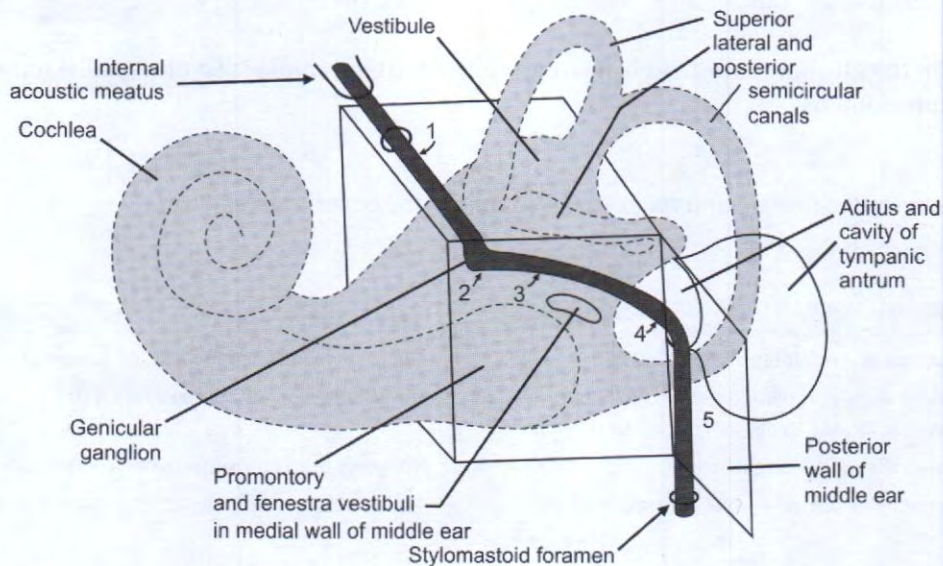


Figure 13: Facial nerve—intrapetrous part

Branches of Distribution

Facial nerve (Intratemporal branches)		
Greater petrosal nerve	Nerve to stapedius	Chorda tympani nerve
<ul style="list-style-type: none"> Arise from geniculate ganglion and enters middle cranial fossa through a hiatus on anterior surface of petrous temporal bone Proceeds towards foramen lacerum, where it joins deep petrosal nerve to form nerve to pterygoid canal Carries gustatory and parasympathetic fibers and supplies lacrimal gland and mucosal glands of nose, palate and pharynx 	<ul style="list-style-type: none"> Arises opposite pyramid of middle ear and supplies stapedius muscle 	<ul style="list-style-type: none"> Arises in vertical part of facial canal about 6 mm above stylomastoid foramen Runs upwards and forwards in a bony canal to enter middle ear and runs forward in close relation to tympanic membrane and exits middle ear by passing through petrotympanic fissure Then it passes medial to spine of sphenoid and enters infratemporal fossa where it joins lingual nerve through which it is distributed Carries preganglionic secretomotor fibers to submandibular and sublingual salivary glands through submandibular ganglion and taste fibers from anterior 2/3rd of tongue

Applied Anatomy

- Injury to facial nerve proximal to origin of chorda tympani leads to loss of taste sensation from anterior 2/3rd of tongue
- Lesion of facial nerve proximal to origin of nerve to stapedius causes hyperacusis (normal sound appear too loud due to loss of dampening effect of stapedius muscle).

11. Otological causes for facial paralysis.

- Facial nerve passes in close relation to middle ear cavity
- Therefore, middle ear pathology can cause facial nerve paralysis.

Otological Causes of Facial Paralysis

<i>Idiopathic</i>	<i>Middle ear</i>					<i>External ear</i>	
	<i>Congenital</i>	<i>Infections</i>	<i>Iatrogenic</i>	<i>Traumatic</i>	<i>Neoplastic</i>	<i>Infections</i>	<i>Neoplastic</i>
Bell's palsy	Congenital cholesteatoma	ASOM CSOM Mastoiditis	Mastoidectomy Stapedectomy Tympanoplasty	Penetrating injury Temporal bone #	Malignancies of middle ear Glomus jugulare	Herpes zoster oticus Malignant otitis externa	Malignancies of external ear

12. BERA (Brainstem evoked response audiometry).

- BERA is audiometric test used to measure tiny physiological electric event occurring in response to sound stimulation by clicks or tone bursts
- A non-invasive, objective audiological investigation introduced by Jewitt (1970)
- Also called ABR (auditory brainstem response), BEP (brainstem evoked potential), AER (auditory evoked response).

Principle

- Sound waves entering cochlea are transduced into electrical potential which are transmitted to relay station, i.e. brain, stem via VIII nerve
- These electrical responses are picked up by surface electrodes and represented graphically
- These graphs contain waves generated by major processing centers of auditory system
- Hearing sensitivity is tested in the range of 1000–4000 Hz.

Procedure

- Examination is done in a comfortable acoustically isolated room with one ear tested at a time
- Place three electrodes as follows:
 - Active electrode On vertex/high forehead
 - Reference electrode On earlobe or mastoid of tested ear
 - Ground electrode On earlobe or mastoid of opposite ear
- Keep ear phone in tested ear
- Provide series of 1000–1200 clicks at a rate of 5–50/sec
- Record neurogenic potentials elicited for first 10 milliseconds (time taken for electrical responses to brainstem alone)
- Repeat the procedure in other ear.

Observation

- Graph plotted shows 7 waves, originating from different centers in auditory pathway

<i>Wave</i>	<i>Site of origin ("E. COLI MA")</i>
I	Cochlear (eighth) nerve
II	Cochlear nuclei (pons)
III	Superior Olivary complex (pons)
IV	Lateral lemniscus (pons)
V	Inferior colliculus (midbrain)
VI	Medial geniculate
VII	Auditory radiation

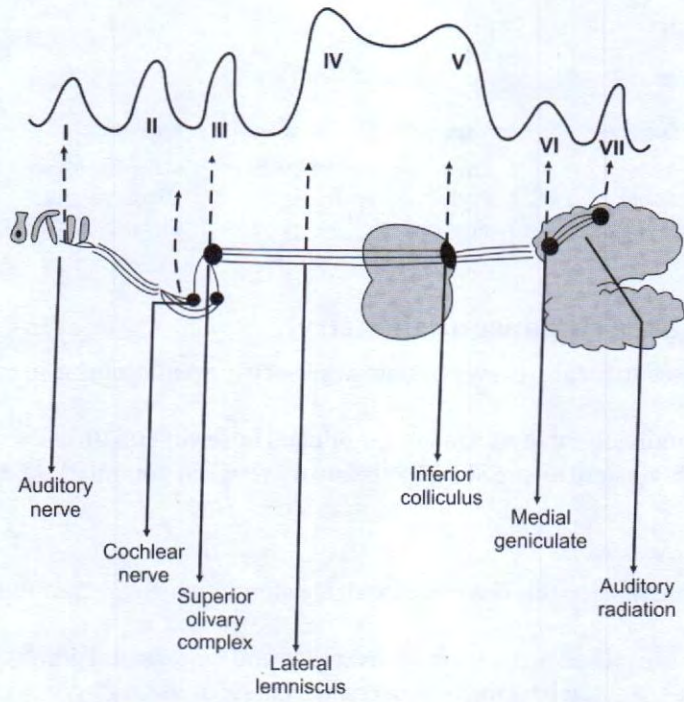


Figure 14: BERA waves—sites of origin

Interpretation

a. Absolute peak latency (in milliseconds)	<ul style="list-style-type: none">♦ From start of click to appearance of a wave♦ Peak latency of wave V is most consistent abnormality
b. Interwave latency delay (in milliseconds)	<ul style="list-style-type: none">♦ Between peaks of two waves♦ Commonly used parameters are I to III, III to V and I to V
c. Wave amplitude (in microvolts μV)	<ul style="list-style-type: none">♦ From peak of a wave to peak of next trough
d. Interaural peak latency (in milliseconds)	<ul style="list-style-type: none">♦ Difference of a particular wave between two ears♦ Only V wave is used as it is most consistent and reliable♦ Used esp. in unilateral ear diseases
e. Absence of expected waves	
f. Deformities of waves	

Normal values

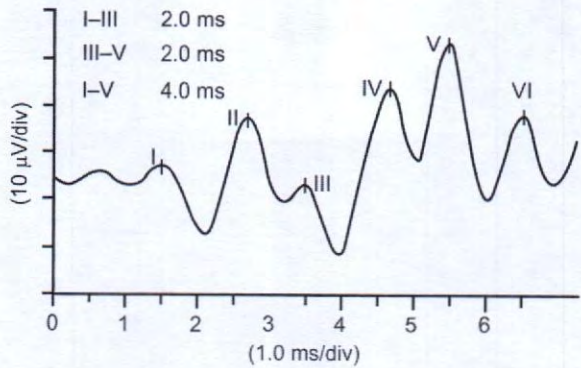


Figure 15: BERA waves—interpretation

- Latency of wave V 5.9 milliseconds
- I-III interval 2.0 milliseconds
- III-V interval 2.0 milliseconds
- I-V interval 4.0 milliseconds
- I-V interaural difference 0.29 millisecond

Applications

- To test integrity of central auditory pathway (as a screening procedures in infants)
- To determine hearing threshold in infants, children and uncooperative adults
- To diagnose retrocochlear pathology specially acoustic neuroma (most reliable investigation)
- To detect malingering
- To assess early neurological brainstem lesions like multiple sclerosis, demyelinating disease, etc.
- To monitor cochlear nerve intraoperatively in surgery of acoustic neuroma to preserve its function.

Advantages	Disadvantages
<ul style="list-style-type: none"> • Can be employed in infants and children • Can be used in cases of mentally handicapped like cerebral palsy, mental retardation and aphasia • Can be used in unconscious patients 	<ul style="list-style-type: none"> • No standardization exists at present • Wave V is not recorded if hearing level is 75 dB at 3 kHz • Latency of wave V normally increases in old age, conductive deafness and pure sensorineural deafness • Wave I not easily identifiable

■ SHORT ANSWERS

13. Cochlear implants.

Refer Question No. 11 June 2012 (RS2).

14. Referred otalgia.

Refer Question No. 3 June 2010 (RS2).

15. Otitis externa.

- Otitis externa is diffuse inflammation of meatal skin which may involve pinna and epidermal layer of tympanic membrane.

Etiology

Causative organisms	Predisposing factors	Portal of entry
<ul style="list-style-type: none"> • <i>Staphylococcus aureus</i> • Streptococci • <i>Pseudomonas pyocyaneus</i> • <i>Bacillus proteus</i> • <i>Escherichia coli</i> 	<ul style="list-style-type: none"> • Hot and humid climate (causes sweating which makes pH of meatal skin alkaline favoring bacterial growth) • Swimmers (favors bacterial invasion through contaminated water) 	<ul style="list-style-type: none"> • Trauma to meatal skin as a result of scratching, unskilled instrumentation, vigorous cleaning, etc. • Spread of infection from otitis media

Phases	Acute phase	Chronic phase
Clinical features		
Symptoms	<ul style="list-style-type: none"> • Hot burning sensation in ear • Pain in ear aggravated by jaw movements and at night (due to congestion in recumbent position) • Ear discharge, thin initially but later thick and purulent • Tinnitus 	<ul style="list-style-type: none"> • Irritation in ear • Strong desire to itch • Scanty discharge

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Phases	Acute phase	Chronic phase
Signs	<ul style="list-style-type: none"> ♦ Inflamed and edematous meatal lining ♦ Conductive hearing loss (due to collection of debris, discharge and meatal swelling) ♦ Tender and enlarged regional lymph nodes ♦ Cellulitis of surrounding tissue 	<ul style="list-style-type: none"> ♦ Thick and swollen meatal skin ♦ Scaling and fissuring of meatal skin ♦ Meatal stenosis due to hypertrophied skin (chronic stenotic otitis externa)
Treatment		
Conservative	<ul style="list-style-type: none"> ♦ Gentle and meticulous ear toilet to remove all exudates and debris by dry mopping, suction clearance or irrigating with warm normal saline ♦ Insertion of gauze wick soaked in antibiotic and steroid and keeping it moist by regular instillation of same drops 2–3 times/day and periodic changing of wick per day for 2–3 days followed by instillation of ear drops ♦ Administration of mild astringents like 8% aluminium acetate or 3% silver nitrate (forms a protective coagulum to dry up oozing meatus) ♦ Broad spectrum antibiotic systemically (in presence of cellulitis or acute tender lymphadenitis) ♦ Analgesic to relieve pain 	<ul style="list-style-type: none"> ♦ 10% ichthymol glycerine soaked gauze wick in ear canal to reduce swelling ♦ Ear toilet ♦ Topical antibiotic steroid cream to control itching
Operative	—	Surgical excision of thickened meatal skin and widening of bony meatus by drill followed by split skin graft

16. Choanal atresia.

Refer Question No. 7 December 2013 (RS2).

17. Rhinolith.

- ♦ Rhinolith is stone formation in nasal cavity.

Etiopathogenesis

Etiology	Pathogenesis
<ul style="list-style-type: none"> ♦ Foreign body (MC) ♦ Blood clot ♦ Inspissated secretions 	<ul style="list-style-type: none"> ♦ Slow deposition of carbonates and phosphates of calcium and magnesium around a nidus

Clinical Features

- ♦ Unilateral.

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Asymptomatic initially when small ♦ Nasal obstruction ♦ Foul smelling, blood stained discharge ♦ Bleeding from nose ♦ Headache and neuralgic pain (due to pressure necrosis of septum and/or lateral wall of nose) 	<ul style="list-style-type: none"> ♦ Grey or greenish black mass with irregular surface near floor of nose ♦ Stony hard and gritty feel on probing ♦ Brittle and may break while manipulating ♦ Surrounded by granulations

Investigations

X-ray	Diagnostic nasal endoscopy
<ul style="list-style-type: none"> ♦ Radio opaque body in nasal cavity 	<ul style="list-style-type: none"> ♦ Confirms location and extent of rhinolith

Treatment*Operative*

- Removal under general anesthesia
 - Through anterior nares for small stones
 - By breaking into smaller pieces for larger stones
 - By lateral rhinotomy for hard and irregular stones.

Complications

- Oroantral fistula.

18. Inverted papilloma.

- Rare benign neoplasm of nose and paranasal sinuses
- So named because neoplastic epithelium grows towards underlying stroma rather than surface
- Also called transitional cell papilloma, Ringertz tumor, Schinzelian papilloma, fungiform papilloma, cylindrical cell papilloma, epithelial papilloma.

<i>Etiology</i>	<i>Site of origin</i>
<ul style="list-style-type: none"> ♦ Idiopathic ♦ Human papilloma virus 6, 11, 16 and 18 (suspected) 	<ul style="list-style-type: none"> ♦ Lateral wall of nose (MC) ♦ Vestibule, septum ♦ Floor of nasopharynx ♦ Sphenoid and frontal sinus ♦ Lacrimal sac

Pathology

<i>Gross appearance</i>	<i>Microscopic appearance</i>
<ul style="list-style-type: none"> ♦ Granular mulberry-like polyp appearance with red to pale pink colour 	<ul style="list-style-type: none"> ♦ Thickened epithelium with extensive invasion into underlying stroma ♦ Covered by alternating layers of squamous and columnar epithelium

Clinical Features

- Mostly seen in males aged 40–70 years
- Always unilateral.

<i>Symptoms</i>	<i>Signs</i>
<ul style="list-style-type: none"> ♦ Nasal obstruction ♦ Facial swelling ♦ Hyponasal speech ♦ Bleeding from nose ♦ Nasal discharge ♦ Decreased or absent smell sensation 	<ul style="list-style-type: none"> ♦ Solitary, granular, firm red or grey mass ♦ Translucent and edematous ♦ Bleeds on touching

Staging (Krouse's staging)

T1	Disease limited to nasal cavity alone
T2	Disease limited to ethmoid sinuses and medial and superior portions of maxillary sinuses
T3	Disease involves lateral or inferior aspects of maxillary sinus or extension into frontal or sphenoid sinuses
T4	Tumor spread outside confines of nose and sinuses (also includes malignancy)

Investigations	Differential diagnosis
a. Radiological <ul style="list-style-type: none"> – X-ray shows involvement of sinuses with associated bone erosions – CT to assess extension of tumor b. Diagnostic nasal endoscopy and biopsy	<ul style="list-style-type: none"> ♦ Antrochoanal polyp ♦ Allergic fungal sinusitis

Treatment

Operative

- Wide surgical excision (complete)

Techniques

- Endoscopic excision using lasers (for small lesions)
- Medial maxillectomy and en bloc ethmoidectomy along with lateral rhinotomy (for larger lesions and malignant tumor).

Prognosis

- Highest recurrence rate (70%)
- Malignant transformation to squamous cell carcinoma (10%).

19. Rhinolalia.

- Change in tonal quality of voice due to nasal or nasopharyngeal problems is called rhinolalia.

Types

- Rhinolalia clausa (hyponasality).
- Rhinolalia aperta (hypernasality).

	<i>Rhinolalia clausa</i>	<i>Rhinolalia aperta</i>
Words affected	Lack of nasal resonance for words which resonate in nasal cavity (m, n, ng)	Nasal resonance of words which have little nasal resonance
Pathogenesis	Because air does not pass through nose due to blockage of nose or nasopharynx	Because of failure of nasopharynx to cut off from oropharynx or abnormal communication between oral and nasal cavities
Etiology	<ul style="list-style-type: none"> ♦ Common cold ♦ Nasal allergy ♦ Nasal polypi ♦ Nasal growth ♦ Adenoids ♦ Nasopharyngeal mass ♦ Familial speech pattern ♦ Habitual 	<ul style="list-style-type: none"> ♦ Valopharyngeal insufficiency ♦ Congenitally short soft palate ♦ Submucous palate ♦ Large nasopharynx ♦ Cleft palate ♦ Paralysis of soft palate ♦ Post adenoidectomy ♦ Post surgery for sleep apnoea ♦ Oronasal fistula ♦ Familial speech pattern ♦ Habitual
Features	<ul style="list-style-type: none"> ♦ Flat dull voice (potato in mouth voice) 	<ul style="list-style-type: none"> ♦ Nasal twang to voice ♦ Nasal regurgitation
Treatment	Treatment of underlying cause	Conservative <ul style="list-style-type: none"> ♦ Speech therapy in mild cases Operative Indications <ul style="list-style-type: none"> ♦ Major defect ♦ Failure of conservative therapy

Contd...

Contd...

<i>Rhinolalia clausa</i>	<i>Rhinolalia aperta</i>
	Techniques <ul style="list-style-type: none"> ♦ Repair of cleft palate using VY plasty to push back palate ♦ Implantation of materials (silastic, Teflon, cartilage) in posterior pharyngeal wall ♦ Pharyngoplasty by pharyngeal flaps to decrease size of nasopharyngeal sphincter

20. Complications of tracheostomy.

Refer Question No. 9 December 2007 (RS2).

21. Palliative treatment.

- Palliative treatment means treatment of cancers with only aim to relieve symptoms and prolong life.

Indications

- Advanced, recurrent carcinoma or distant metastases
- Poor general condition of patient to undergo surgery.

Modalities

<i>Chemotherapy</i>	<i>Radiation</i>	<i>Surgery</i>
Use of single or multiple drugs with minimum toxicity	Use of radiation to minimum possible	Debulking of tumor mass

22. Chemotherapy.

- Chemotherapy is modality of cancer of treatment using antineoplastic drugs either alone or in combination.

Principle

- Weakening or destruction of cancer cells at primary and metastatic site by interfering with stages of cell cycle.

Types

<i>Adjuvant</i>	<i>Palliative</i>	
Use of drugs before, during or after treatment with other modalities like surgery or radiation	Use of drugs only to relieve symptoms and prolong life in cases of advanced, recurrent or metastatic cancer	
↓	↓	↓
Induction (anterior chemotherapy)	Concurrent	Posterior chemotherapy
Chemotherapy before surgery or radiation to reduce tumor burden and prevent micrometastases	Chemotherapy along with radiation to act as radiosensitizer to radioresistant tumors	Chemotherapy after surgery or radiation to treat micrometastases occurring during surgery or radiation

Drugs Commonly Used

<ul style="list-style-type: none"> ♦ Methotrexate ♦ 5-Fluorouracil ♦ Cyclophosphamide 	<ul style="list-style-type: none"> ♦ Dacarbazine ♦ Bleomycin ♦ Actinomycin 	<ul style="list-style-type: none"> ♦ Vincristine ♦ Cisplatin
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<i>Advantages</i>	<i>Disadvantages</i>
<ul style="list-style-type: none"> ♦ Provides placebo effect in patients with advanced cancers 	<ul style="list-style-type: none"> ♦ Highly toxic because affection of normal dividing cells causing alopecia, stomatitis, nausea, vomiting, diarrhea, bone marrow depression, decreased blood cells, etc.

MBBS PHASE III EXAMINATION

JUNE 2009

(Revised Scheme 2)

LONG ESSAYS

1. Discuss the etiopathology, clinical features and management of epistaxis.

- Epistaxis is bleeding from inside of nose.

Etiology

Local	Systemic	Idiopathic
A. Nose a. Congenital <ul style="list-style-type: none"> – Multiple telangiectases (Osler's disease) b. Trauma <ul style="list-style-type: none"> – Fingernail trauma – Injuries of nose – Intranasal surgery – Fracture of middle 1/3rd of face and base of skull – Hard blowing of nose – Violent sneeze c. Infections <ul style="list-style-type: none"> i. Acute <ul style="list-style-type: none"> – Viral rhinitis – Nasal diphtheria – Acute sinusitis ii. Chronic (all crust forming diseases) <ul style="list-style-type: none"> – Atrophic rhinitis – Rhinitis sicca – Tuberculosis – Syphilis – Rhinosporidiosis d. Foreign bodies <ul style="list-style-type: none"> i. Non-living <ul style="list-style-type: none"> – Rhinolith ii. Living <ul style="list-style-type: none"> – Maggots e. Tumors of nose and PNS <ul style="list-style-type: none"> i. Benign <ul style="list-style-type: none"> – Hemangioma – Papilloma ii. Malignant <ul style="list-style-type: none"> – Carcinoma – Sarcoma f. Atmospheric pressure changes <ul style="list-style-type: none"> – High altitude – Sudden decompression g. Deviated nasal septum	a. Cardiovascular system <ul style="list-style-type: none"> – Hypertension – Arteriosclerosis – Mitral stenosis – Pregnancy b. Blood and blood vessels disorders <ul style="list-style-type: none"> – Aplastic anemia – Leukemia – Thrombocytopenic purpura – Vascular purpura – Hemophilia – Christmas disease – Scurvy – Vitamin K deficiency c. Liver diseases <ul style="list-style-type: none"> – Cirrhosis of liver d. Renal diseases <ul style="list-style-type: none"> – Chronic nephritis e. Drugs (excessive use of) <ul style="list-style-type: none"> – Salicylates – Anticoagulants f. Mediastinal compression (venous pressure in nose) <ul style="list-style-type: none"> – Tumors of mediastinum g. Acute general infections <ul style="list-style-type: none"> – Influenza – Measles – Chickenpox – Whooping cough – Rheumatic fever – Infectious mononucleosis – Typhoid – Pneumonia – Malaria – Dengue h. Vicarious menstruation (epistaxis at time of menstruation)	
B. Nasopharynx <ul style="list-style-type: none"> – Adenoiditis – Juvenile angiofibroma – Malignant tumors 		

Classification

<i>Anterior epistaxis</i>	<i>Posterior epistaxis</i>
<ul style="list-style-type: none"> ♦ More common form and seen usually in children and young adults ♦ Blood flows out from front of nose with patient in sitting position ♦ Caused by trauma ♦ Usual site of bleeding is from Little's area or anterior part of lateral wall ♦ Bleeding is mild and can be controlled by local pressure or anterior pack 	<ul style="list-style-type: none"> ♦ Less common and seen only in adults above 40 years of age ♦ Blood flows back into throat which patient swallows and later vomits as coffee colored vomitus ♦ Occurs spontaneously due to hypertension or arteriosclerosis ♦ Severe bleeding occurs mostly from posterosuperior part of nasal cavity ♦ Requires hospitalization and postnasal packing

Clinical Features

<i>Symptoms</i>	<i>Signs</i>
<p>a. Bleeding</p> <ul style="list-style-type: none"> – Vary quantity of blood (trivial to profuse) may be seen bleeding from nose (anterior epistaxis) or may be swallowed by patient (posterior epistaxis) – May be continuous or intermittent 	<p>a. Anxiety</p> <ul style="list-style-type: none"> – Due to sight of blood <p>b. Hemoptysis or hematemesis</p> <ul style="list-style-type: none"> – Swallowed blood may be brought out in sputum or vomitus <p>c. Shock</p> <ul style="list-style-type: none"> – In case of severe bleeding

Investigations

a. Blood pressure	<ul style="list-style-type: none"> ♦ Elevated in hypertension ♦ Hypotension is indicative of massive blood loss
b. Hb%	<ul style="list-style-type: none"> ♦ Assesses degree of anemia ♦ Helps to calculate requirement of blood for transfusion
c. Coagulation tests	<ul style="list-style-type: none"> ♦ May be abnormal in bleeding disorders or coagulopathies
d. Radiography	<ul style="list-style-type: none"> ♦ X-ray skull to detect acute sinusitis, fracture or malignancy of PNS ♦ CT scan
e. Endoscopy (of nose and PNS)	<ul style="list-style-type: none"> ♦ To detect site of bleeding
f. Biopsy	<ul style="list-style-type: none"> ♦ To reveal nature of lesion

Treatment

<i>Supportive</i>	<i>First aid</i>
<ul style="list-style-type: none"> ♦ Make patient sit up with back rest ♦ Record any blood loss through spitting or vomiting ♦ Reassurance and mild sedation in anxious patients ♦ Monitor pulse, BP and respiration ♦ Maintain hemodynamic and transfuse blood if required ♦ Antibiotics if nasal packing kept for more than 24 hours (to prevent infections) ♦ Intermittent oxygen in patients with bilateral nasal packing (because of increased pulmonary resistance from nasopulmonary reflex) 	<p>a. Pinching of nose</p> <ul style="list-style-type: none"> – Pinching of nose with thumb and index finger for 5 minutes easily controls bleeding from Little's area <p>b. Trotter's method</p> <ul style="list-style-type: none"> – Make patient to sit and lean little forward over a basin to spit any blood and breath quietly from mouth – He is not allowed to swallow blood but asked to spit it out <p>c. Cold compress</p> <ul style="list-style-type: none"> – Cold compress with ice over nose causes reflex vasoconstriction

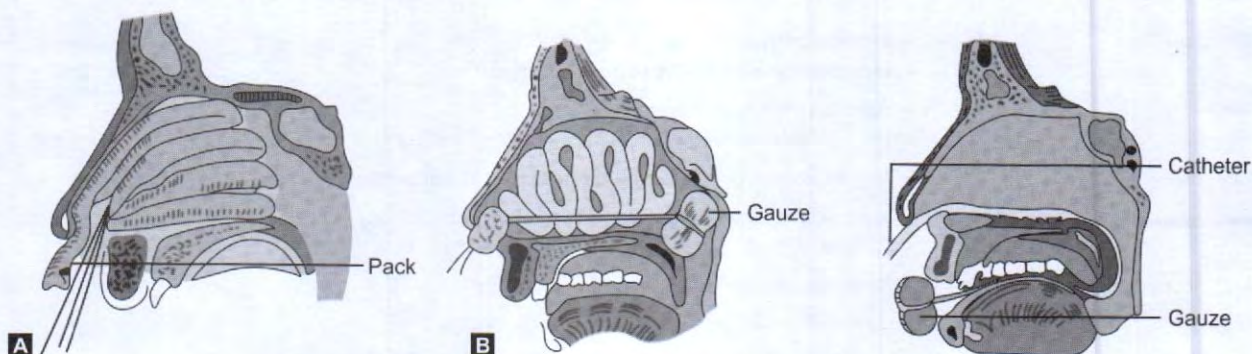
Conservative (local treatment)

a. Cauterization

	<i>Direct</i>	<i>Endoscopic</i>
Indications	<ul style="list-style-type: none"> ♦ Anterior epistaxis with accessible bleeding point 	<ul style="list-style-type: none"> ♦ Posterior epistaxis
Procedure	<ul style="list-style-type: none"> ♦ After local anesthesia using xylocaine pack, bleeding point is cauterized with a bead of 15% silver nitrate (chemical cautery) or coagulated with electrocautery 	<ul style="list-style-type: none"> ♦ After local anesthesia and sedation, bleeding point is located using nasal endoscope and bleeding point is coagulated with electrocautery

b. Nasal packing

	<i>Anterior nasal packing</i>	<i>Posterior nasal packing</i>
Indications	<ul style="list-style-type: none"> ♦ Active anterior epistaxis (profuse bleeding from inaccessible site) 	<ul style="list-style-type: none"> ♦ Posterior epistaxis ♦ Continuous bleeding inspite of anterior nasal packing
Anesthesia	<ul style="list-style-type: none"> ♦ Local anesthesia 	<ul style="list-style-type: none"> ♦ General anesthesia ♦ Local anesthesia with sedation
Procedure	<ul style="list-style-type: none"> ♦ Clear nose of blood clot by suction ♦ Soak 1 meter gauze (2.5 cm wide in adults and 12 mm in children) in liquid paraffin (to avoid sticking to nasal mucosa otherwise its removal becomes difficult, painful resulting in fresh bleeding) ♦ Fold first few centimeters of gauze upon itself and insert along floor ♦ Then tightly pack entire nasal cavity by layering gauze from floor to roof from before backwards ♦ One or both cavities can be packed ♦ If bleeding stops, pack can be removed after 24 hours ♦ If pack need to kept for 2–3 days then prophylactic antibiotics should be administered to prevent sinus infection and toxic shock syndrome 	<ul style="list-style-type: none"> ♦ Prepare postnasal pack by tying 3 silk threads to a piece of gauze rolled into shape of cone ♦ Pass a rubber catheter through nose and bring its end out from mouth ♦ Tie 2 ends of silk thread to it and withdraw catheter from nose ♦ Guide postnasal pack into nasopharynx with index finger and make sure its fitted there snugly ♦ Tie threads in front of nasal columella after protecting if with a piece of gauze ♦ Pack anterior nasal cavity by tying one of the silk thread over a dental roll ♦ Cut short third silk thread, allowing it to hang in oropharynx (for easy removal of pack later)



Figures 1A and B: (A) Anterior nasal packing; (B) Posterior nasal packing

Alternatives

- Bulb of Foley's catheter passed through nose is inflated using saline and pulled back to block choana and then anterior pack is kept in usual manner
- Brighton nasal balloons with two bulb are available, one fixed balloon for postnasal space and other sliding anterior balloon for nasal cavity.

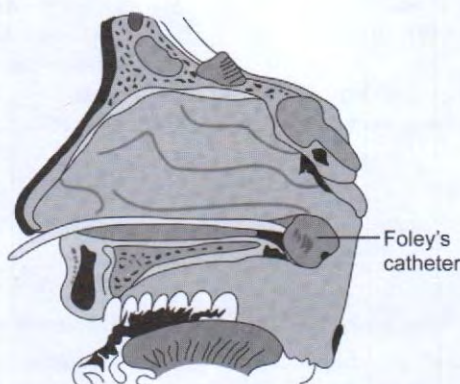


Figure 2: Posterior nasal packing with Foley's catheter

Operative

	Elevation of mucoperichondrial flap and SMR operation	Ligation or embolization of blood vessels
Indications	<ul style="list-style-type: none"> ♦ Persistent or recurrent bleeding from septum 	<ul style="list-style-type: none"> ♦ Failure of conservative treatment ♦ Recurrent epistaxis
Techniques	<ul style="list-style-type: none"> ♦ Involves elevation of mucoperichondrial flap and reposition back to cause fibrosis and constriction of blood vessels 	<ul style="list-style-type: none"> i. External carotid artery <ul style="list-style-type: none"> – Ligation of external carotid above origin of superior thyroid artery ii. Maxillary artery <ul style="list-style-type: none"> – Blocking of maxillary artery or its branches using clips via Caldwell-Luc approach or endoscope iii. Ethmoidal arteries <ul style="list-style-type: none"> – Ligation of anterior and posterior ethmoidal arteries in medial wall of orbit through external ethmoidal incision

Specific

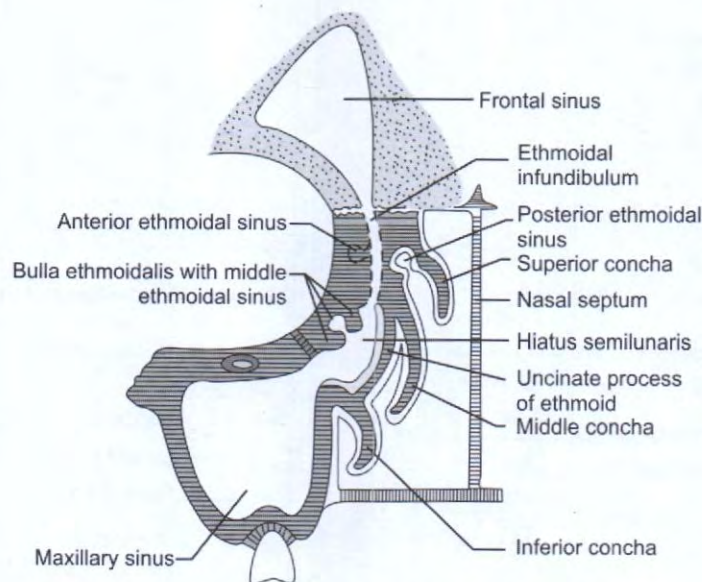
- Treatment of underlying cause.

2. Describe the anatomy and functions of paranasal sinuses.

- Paranasal air sinuses are air filled spaces present within cranial bones around nasal cavities
- They are arranged in pairs and all of them open into nasal cavity
- They are absent at birth but enlarge rapidly with growth of bone.

Classification

- Frontal
- Maxillary
- Sphenoidal
- Ethmoidal.

**Figure 3:** Paranasal air sinuses—location and drainage**A. Frontal sinus**

- Frontal air sinuses are paired paranasal sinuses.

Location

- Squamous part of frontal bone deep to medial end of superciliary arch extending upwards above medial end of eyebrow and backward into medial part of roof of orbit.

Measurements

- Triangular in shape
- Vertical: 3 cm
- Transverse: 2.5 cm
- Anteroposterior: 1.8 cm.

Drainage

- Into middle meatus of nose at anterior end of hiatus semilunaris either through infundibulum or frontonasal duct.

Arterial supply

- Supraorbital artery.

Venous drainage

- Into anastomotic vein between supraorbital and superior ophthalmic veins.

Lymphatic drainage

- Submandibular nodes.

Nerve supply

- Supraorbital nerve.

Development

- Rudimentary or absent at birth and development start only after 2-3 years after birth
- Well developed by 7-8 years of age but reach full size only after puberty.

Applied anatomy

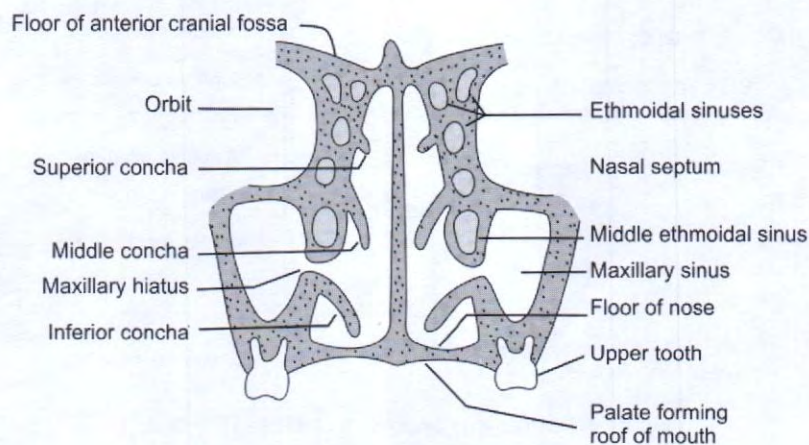
- Infection of frontal sinus is called frontal sinusitis manifesting as headache and persistent, thick and purulent nasal discharge
- May cause complications like brain abscess in frontal lobe.

B. Maxillary sinus

- Largest paranasal sinuses
- First sinus to develop.

Location

- Body of maxilla.

**Figure 4: Maxillary sinus***Measurements*

- Pyramidal in shape
- Height: 3.5 cm

- Width: 2.5 cm
- Anteroposterior depth: 3.5 cm.

Features

- a. Apex
 - Formed by zygomatic process of maxilla.
- b. Base
 - Formed by nasal surface of body of maxilla
 - Directed medially towards lateral wall of nose
 - In disarticulated skull, base presents a large opening, maxillary hiatus, which is reduced in size by following bones
 - * Superiorly : Uncinate process of ethmoid bone
 - * Inferiorly : Ethmoidal process of inferior nasal concha
 - * Anteriorly : Descending process of lacrimal bone
 - * Posteriorly : Perpendicular plate of palatine bone
 - It is further reduced in size by thick mucosa of nose.
- c. Roof
 - Formed by orbital surface of maxilla
 - Traversed by infraorbital vessels and nerve in a bony canal.
- d. Floor
 - Formed by alveolar process of maxilla
 - Lies about 1 cm below level of level of floor of nose which corresponds to level of lower border of ala of nose
 - Floor is marked by several conical elevations produced by roots of upper molar and premolar teeth and roots may even penetrate bony floor to lie beneath mucus lining.
- e. Anterior wall
 - Related to infraorbital plexus of vessels and nerves and origins of muscles of upper lip
 - Within anterior wall, anterior superior alveolar vessels and nerves traverse in a bony canal, the canalis sinus.
- f. Posterior wall
 - Pierced by posterior superior alveolar vessels
 - Forms anterior boundary of infratemporal and pterygopalatine fossa.

Drainage

- Into middle meatus through floor of hiatus semilunaris and opening lies just below bulla ethmoidalis
- Opening is located much higher than floor of sinus.

Relations

- Superiorly : Zygomatic process and orbital surface of maxilla
- Inferiorly : Nasal surface of body of maxilla
Roots of upper molar and premolar teeth
- Anteriorly : Infraorbital plexus of vessels and nerves
Origins of muscles of upper lip
- Posteriorly : Infratemporal and pterygopalatine fossa

Arterial supply

- Facial, infraorbital and greater palatine arteries.

Venous drainage

- Into facial vein and pterygoid plexus of veins.

Lymphatic drainage

- Into submandibular nodes.

Nerve supply

- Infraorbital and anterior, middle and posterior superior alveolar nerves.

Applied anatomy

- Maxillary sinus is most common paranasal sinus to get infected
- Carcinoma of maxillary sinus arises from mucosal lining.

C. Sphenoidal sinus

- Sphenoidal air sinuses are 2 paired asymmetrical sinuses.

Location

- In body of sphenoid bone above and behind nasal cavity.

Boundaries

- Anterior : Roof of the orbit
- Posterior : Anterior margin of foramen magnum
- Lateral : Pterygoid canal.

Drainage

- Into sphenoethmoidal recess above superior turbinate.

Relations

- Laterally Cavernous sinus containing third, fourth, fifth and sixth cranial nerves
Internal carotid artery
Optic nerve
- Superiorly Hypophysis cerebri
Optic chiasma
Frontal lobe of cerebrum
Olfactory bulb
- Inferiorly Roof of nasopharynx
- Posteriorly Brainstem
Basilar artery
- Anteriorly Nasal cavity (sphenoethmoidal recess)

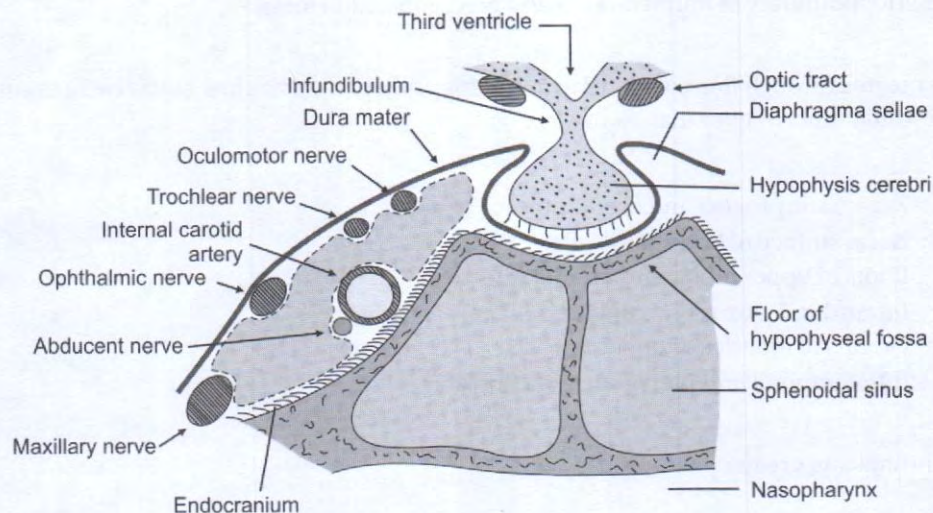


Figure 5: Sphenoidal sinus—relations

Arterial supply

- Posterior ethmoidal and internal carotid arteries.

Venous drainage

- Into pterygoid venous plexus and cavernous sinus.

Lymphatic drainage

- Retropharyngeal nodes.

Nerve supply

- Posterior ethmoidal nerve and orbital branches of pterygopalatine ganglion.

Applied anatomy

- Hypophysis cerebri can be approached through sphenoidal air sinus by transantral or transnasal routes.

D. Ethmoidal sinuses

- Ethmoidal sinuses are numerous small intercommunicating spaces in cranium.

Location

- Within labyrinth or lateral part of ethmoid bone.

Boundaries

- Superiorly: Orbital plate of frontal bone
- Anteriorly: Lacrimal bone
- Posteriorly: Sphenoidal conchae and orbital process of palatine bone.

Classification

- Anterior group
- Middle group
- Posterior group.

Features

Features	Anterior ethmoidal sinus	Middle ethmoidal sinus	Posterior ethmoidal sinus
♦ Air cells	1–11	1–7	1–7
♦ Drainage	Anterior part of hiatus semilunaris or Ethmoidal infundibulum	On or near bulla ethmoidalis in middle meatus of nose	Superior meatus of nose
♦ Arterial supply	Anterior ethmoidal artery	Posterior ethmoidal artery	Posterior ethmoidal artery
♦ Nerve supply	Anterior ethmoidal nerve	Posterior ethmoidal nerve Orbital branches of pterygopalatine ganglion	Posterior ethmoidal nerve Orbital branches of pterygopalatine ganglion
♦ Lymphatics	Submandibular nodes	Submandibular nodes	Retropharyngeal nodes

Functions of Paranasal Air Sinuses

- Air contained in paranasal sinuses adds humidity and temperature to inspired air and thus serves as air conditioning chambers
- They act as resonating chambers for production of sounds
- They make facial bone lighter and establish adult contour of face.

SHORT ESSAYS**3. Vestibular function tests.**

- Assessment of vestibular function can be done in two ways
 - Clinical tests
 - Laboratory tests.
- A. Clinical tests
- a. Spontaneous nystagmus
 - Nystagmus is involuntary, rhythmical, oscillatory movements of eye
 - Is an important sign in evaluation of vestibular system.

Types

- Central (due to lesion in vestibular nuclei, brainstem or cerebellum)
- Peripheral (due to lesion of labyrinth or VIII nerve).

Procedure

- With patient seated front of examiner or lying supine on bed, keep examiner's finger 30 cm from patient's eye in central position and move it to right, left, up or down $<30^\circ$ from central position.

Inference

- Nystagmus to side of lesion due to irritative lesion of labyrinth (serous labyrinthitis)
- Nystagmus to healthy side due to parietic lesions of labyrinth (purulent labyrinthitis, labyrinthine trauma, VIII nerve section).

Degree of nystagmus

- 1st degree : Weak nystagmus, present when patient looks in direction of fast component
- 2nd degree : Stronger than 1st degree, present when patient looks straight ahead
- 3rd degree : Stronger than 2nd degree, present even when patient looks in direction of slow component.

b. Fistula test

Principle

- Induction of nystagmus due to transmission of altering pressure in external canal to labyrinth.

Procedure

- Apply intermittent pressure in external ear by pressing on tragus or using Siegle's speculum.

Observation

- Observe for giddiness, nausea or vomiting with or without nystagmus
- Eye deviates towards opposite side with fast component of nystagmus towards same side.

Inference

- Absence of nystagmus (negative test)	Normal
- Presence of nystagmus (positive test)	Erosion of horizontal semicircular canal (cholesteatoma), fenestration operation, post stapedectomy fistula or rupture of round window membrane

Fallacies

- False negative fistula test
 - * Indicates presence of fistula but dead labyrinth or blocking fistula by cholesteatoma or granulations
- False positive fistula test
 - * Indicates no fistula but transmission of air pressure to labyrinth due to hypermobile footplate of stapes (congenital syphilis, post stapedectomy, Meniere's disease).

Significance

- Fistula tests helps to assess functioning of labyrinthine (positive fistula test implies functioning labyrinthine)
- It diagnoses labyrinthine fistula.

c. Romberg's test

Procedure

- Ask patient to stand feet together and arms by side with eyes first open and then closed.

Observation

- Observe for patient's swaying from side to side.

Inference

- Stability	Normal (perform sharpened Romberg test)
- Swaying in different directions	Central vestibular lesions
- Swaying (to side of lesion)	Peripheral vestibular lesion

d. Sharpened Romberg test

Procedure

- Ask patient to stand with one heel in front of toes and arms folded across chest

Inference

- Stability Normal
- Instability Vestibular impairment

e. Gait test

Procedure

- Ask patient to walk along a straight line to a fixed point, with eye open and then eye closed.

Inference

- Deviation with eyes closed (to affected side) Uncompensated lesion of peripheral vestibular system

f. Unterberger's test

Procedure

- Ask patient to march on spot inside a circle with closed eyes and hands outstretched.

Inference

- Deviation (to affected side) Unilateral paralytic labyrinthitis

g. Past pointing and falling

Principle

- Patient voluntarily compensates for sensation of object turning away from him by touching a fixed object and similarly corrects hallucination of movements by falling in opposite direction.

h. Hallpike maneuver (Positional test)

Indications

- Vertigo in certain head position

Procedure

- Make patient sit on couch with examiner holding head of patient
- Turn patient's head to 45° to right and place him in supine position with head hanging 30° below horizontal
- Repeat procedure with head of patient turned to left and finally in straight head hanging position.

Observation

- Observe nystagmus for latency, duration, direction and fatigability

Inference

Latency	Duration	Direction	Fatigability	Diagnosis
2-20second	<1 minute	Always towards undermost ear	Yes	Benign paroxysmal positional vertigo
Nil	As long as head is in critical position	Variable with test position	No	Central lesion (tumors of IV ventricle, cerebellum, temporal lobe, multiple sclerosis, raised intracranial tension)

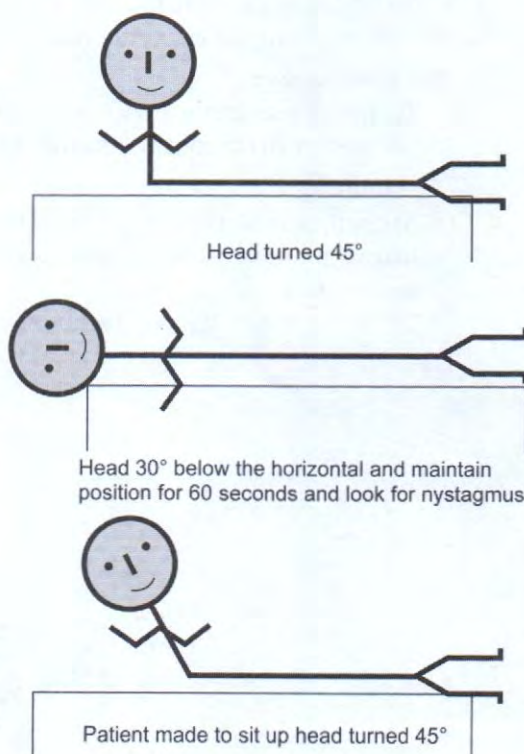


Figure 6: Hallpike maneuver

B. Laboratory tests

a. Caloric test

Principle

- Assessment of nystagmus induced by thermal stimulation of vestibular system

- Hot water heats fluid in duct, lowering its specific gravity and raising its level thus it moves towards ampulla causing nystagmus with quick component to simulated side
- Similarly cold water produces nystagmus in opposite direction.

Methods

i. Modified Kobrak test (cold tap water test)

Procedure

- Ask patient to sit with head tilted 60° backwards such that horizontal canal come in vertical position
- Irrigate each ear with ice water for 1 minute, starting with 5 mL and gradually raising to 10 mL, 20 mL and 40 mL if there is no response.

Inference

- Nystagmus with 5 mL Normal
- Nystagmus with 5–40 mL Hypoactive labyrinth
- No nystagmus with 40 mL Dead labyrinth

ii. Fitzgerald—Hallpike test (bithermal caloric test)

Contraindications

- Tympanic membrane perforations (cold air caloric test preferred)
- Severe vertigo (should be controlled first).

Procedure

- Ask patient to lie supine with head tilted 30° forwards such that horizontal canal comes in vertical position
- Irrigate alternate ears for 40 seconds each with water at 30°C and 44°C (7°C on either side of normal body temperature)
- Use water at 20°C if no nystagmus is elicited from any ear.

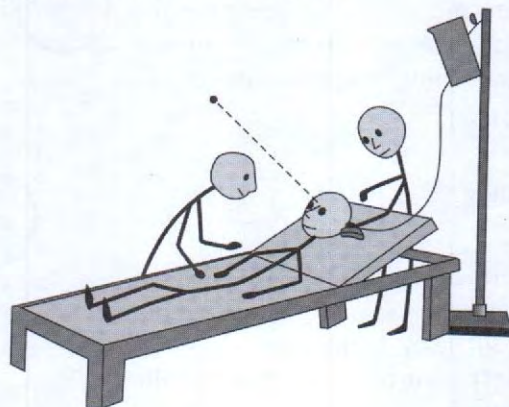


Figure 7: Fitzgerald—hallipike test (Calorie test)

Observation (Cold water induced nystagmus in opposite eye and warm water in same eye)

- Observe eyes for nystagmus from start of irrigation to end point and time taken is recorded on calorigram as follows:
 - * L_{30} and R_{30} = Response to water at 30°C by both left and right ears respectively
 - * L_{44} and R_{44} = Response to water at 44°C by both left and right ears respectively.
- Normally time ranges from 1 minute 20 seconds to 2 minutes

Inference

- Nystagmus with 30°C and 44°C water Normal
- Nystagmus with 20°C water Hypoactive labyrinth
- No nystagmus with 20°C water Dead labyrinth

iii. Cold air caloric test (Dundas Grant's test)

- Rough qualitative test.

Indications

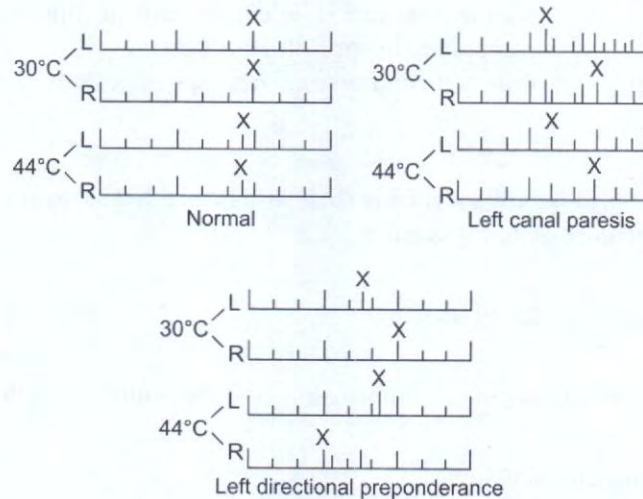
- Perforated tympanic membrane.

Procedure

- Cool air is delivered into ear using Dundas Grant tube (coiled copper tube wrapped in cloth) and ethyl chloride to cool air in tube.

Significance

- Each labyrinth can be tested separately
- Diagnoses vertigo of labyrinthine origin
- Diagnoses canal paresis or dead labyrinth and calculates direction preponderance as follows:

**Figure 8: Calorigram****i. Canal paresis**

- Indicates less response from a particular canal in comparison to opposite side
- Expressed as percent of total response from both ears

$$\text{Response from left ear} = \frac{L_{30} + L_{44}}{L_{30} + L_{44} + R_{30} + R_{44}} \times 100$$

$$\text{Response from right ear} = \frac{R_{30} + R_{44}}{L_{30} + L_{44} + R_{30} + R_{44}} \times 100$$

- Canal paresis is seen in Meniere's disease, acoustic neuroma, post labyrinthectomy or vestibular nerve section.

ii. Direction preponderance

- 25-30% or more increased nystagmus on one side than opposite side is termed direction preponderance
- Considers duration of nystagmus on right or left side irrespective of its origin

$$\text{Right beating nystagmus} = \frac{L_{30} + R_{44}}{L_{30} + L_{44} + R_{30} + R_{44}} \times 100$$

$$\text{Left beating nystagmus} = \frac{R_{30} + L_{44}}{L_{30} + L_{44} + R_{30} + R_{44}} \times 100$$

- Direction preponderance occurs towards side of central lesion and away from side of peripheral lesion
- Directional preponderance along with canal paresis on opposite side indicates unilateral Meniere's disease
- Directional preponderance along with canal paresis on same side indicates acoustic neuroma.

b. Electronystagmography

- Involves recording of corneoretinal potential by placing electrodes at suitable places around eye to detect nystagmus undetectable by naked eye.

Advantages

- Provides permanent record of nystagmus.

c. Optokinetic tests

Procedure

- Ask patient to follow a series of vertical stripes on a drum moving first from right to left and then from left to right.

Inference

- Normally Nystagmus with slow component in direction of moving stripes and fast component in opposite direction
- Optokinetic abnormalities Brainstem and cerebral hemisphere lesions

d. Rotation test

Procedure

- Rotate a patient to seated in Barany's revolving chair with head tilted 30° forwards at rate of 10 turns in 20 secs
- Stop chair abruptly and observe for nystagmus.

Inference

- Normally Nystagmus for 25–40 secs

Advantages

- Can be performed in cases of congenital abnormalities (where caloric test is not possible).

Disadvantages

- Labyrinths cannot be tested individually.

e. Galvanic test

Procedure

- Pass 1 mA current through one ear of patient standing with feet together, eye closed and arms outstretched.

Inference

- Normally, person sways towards side of anodal current.

Advantages

- Only test to differentiate end organ lesion from vestibular nerve lesion.

f. Posturography

- Measuring of postural stability to evaluate vestibular function by using fixed or moving platforms.

4. Lateral sinus thrombosis.

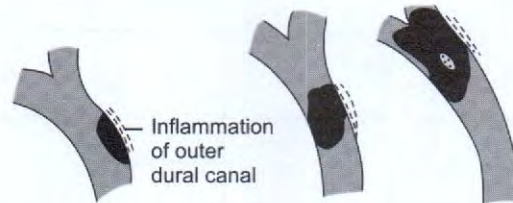
- Lateral sinus thrombosis is inflammation of inner wall of lateral venous sinus with formation of thrombus
- It is an intracranial complication of suppurative otitis media
- Also called sigmoid sinus thrombosis.

Etiology

<i>Causative organisms</i>	<i>Predisposing factors (complication of)</i>	<i>Spread of infection</i>
<ul style="list-style-type: none"> ♦ <i>Staphylococcus haemolyticus</i> ♦ <i>Bacillus proteus</i> ♦ <i>Pseudomonas pyocyaneus</i> ♦ <i>E. coli</i> ♦ Hemolytic streptococci ♦ <i>Pneumococcus</i> 	<ul style="list-style-type: none"> ♦ Acute coalescent mastoiditis ♦ Masked mastoiditis ♦ CSOM ♦ Cholesteatoma 	<ul style="list-style-type: none"> ♦ Erosion of sinus plate <ul style="list-style-type: none"> – Due to hyperemic decalcification or osteitis in acute infections – Due to cholesteatoma or granulation tissue in chronic infections ♦ Retrograde thrombophlebitis ♦ Infection of mastoid bone is transmitted through → Veins of Haversian canals → Dural veins → Dural venous sinuses → Thrombophlebitis of venous sinuses and cortical vein thrombosis

Pathogenesis (Stages)

<i>Formation of perisinus abscess</i>	<i>Endophlebitis and mural thrombus formation</i>	<i>Obliteration of sinus lumen and intrasinus abscess</i>	<i>Extension of thrombus</i>
Destruction of overlying bony dural plate by coalescent bone erosion or cholesteatoma resulting in formation of abscess in relation to outer wall of sinus.	Spread of inflammation to inner wall of venous sinus with deposition of fibrin, platelets and blood cells leads to thrombus formation.	Enlarging thrombus occludes lumen of sinus and secondary invasion by microorganisms leads to intrasinus abscess which releases infected emboli in blood stream causing septicemia.	Intrasinus abscess breaks central part of thrombus but thrombotic process continues proximally spreading to confluence of sinuses, superior sagittal sinus or cavernous sinus and distally to involve mastoid emissary vein, jugular bulb or jugular vein.

**Figure 9:** Progression of venous sinus thrombophlebitis**Clinical Features**

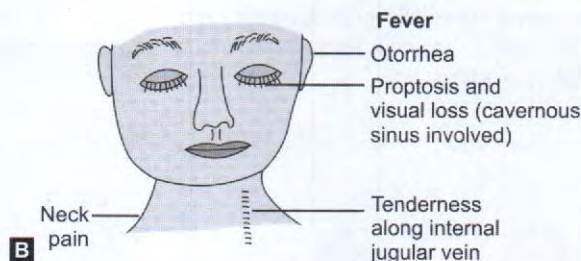
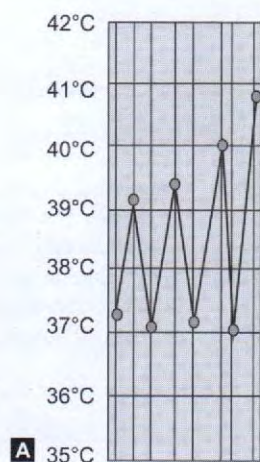
- History of ear disease

Symptoms

- Hectic fevers with rigor
 - Severe fever, irregular in frequency with daily 1 or more peaks (Picket-fence type of fever)
 - Accompanied by chills and rigor
 - Peak followed by fall in temperature with profuse sweating
 - Patient apparently normal between peaks of fever
 - Coincides with release of septic emboli in blood stream
- Headache
 - Mild initially due to perisinus abscess
 - Severe later due to venous stasis raising intracranial pressure
- Projectile vomiting and neck rigidity
 - Indicates raised intracranial pressure and meningeal irritation

Signs

- Progressive anemia and emaciation
- Tenderness along jugular vein
 - Due to extension of thrombophlebitis along jugular vein
 - Associated with enlargement and inflammation of jugular lymph nodes and torticollis
- Greisinger's sign
 - Edema over posterior part of mastoid
- Papilloedema
 - Due to thrombosis of mastoid emissary vein
 - Blurring of disc margins, retinal hemorrhages or dilated retinal veins on ophthalmoscopy
 - Due to obstruction of venous return by thrombosis especially of right sinus
- Tobey-Ayer test
 - No rise in CSF pressure on manual compression of jugular vein on thrombosed side
- Crowe-Beck test (Lillie-Crowe sign)
 - Engorgement of retinal veins and supraorbital veins on compression of jugular vein on healthy side



Figures 10A and B: (A) Sigmoid thrombophlebitis with hectic temperature; (B) Lateral sinus thrombophlebitis: clinical features

Investigations

Blood investigations	Ear swab	CSF examination	Radiology
<ul style="list-style-type: none"> Hemogram reveals anemia Cell count reveals leukocytosis Blood smear to rule out malaria Blood culture and sensitivity to find causative organism 	<ul style="list-style-type: none"> Culture and sensitivity of ear discharge 	<ul style="list-style-type: none"> To exclude meningitis 	<ul style="list-style-type: none"> X-ray mastoid shows clouding of air cells (acute mastoiditis) or bony destruction (cholesteatoma) Contrast CT scan reveals delta sign (triangular area with rim enhancement of sinus wall and central low density area in posterior cranial fossa) MRI better delineates thrombus, demonstrates delta sign on contrast studies and helps assess progression and resolution of thrombus on venography

Treatment

Supportive	Specific	
	Conservative	Operative
a. Repeated blood transfusion – To correct anemia and improve immune resistance	a. Antibiotics – High doses of IV antibiotics depending upon sensitivity results continued till 1 week after surgery – Usually used are combinations 2 or more antibiotics like ampicillin, chloramphenicol, cephalosporins or aminoglycosides b. Anticoagulant therapy – Indicated if thrombosis involves cavernous sinus	a. Mastoidectomy and exposure of sinus <i>Indications</i> – No improvement within 48 hours with conservative therapy <i>Technique</i> – Exposure of sinus by cortical or modified radical mastoidectomy followed by – Drainage of perisinus abscess, intrasinus abscess – Removal of infected clot – Incision of diseased dura mater b. Ligation of internal jugular vein <i>Indications</i> – Failure of conservative and operative treatment – Involvement of jugular vein

Complications (Spread of Infection)

- Septicemia and pyemic abscesses in lung, bone, joints or subcutaneous tissue due to blood borne spread
- Meningitis and subdural abscess due to spread of infection through inner wall
- Cerebellar abscess
- Thrombosis of jugular bulb and jugular vein along with involvement of 9th, 10th and 11th cranial nerve due to distal spread
- Cavernous sinus thrombosis due to proximal spread
- Otitic hydrocephalus.

5. Adult and infantile larynx.

- Larynx is made up of cartilaginous framework lying in front of hypopharynx.

Comparison of Adult and Infantile Larynx

Points of comparison	Infant	Adult
a. Size	Smaller	Larger
b. Shape	Funnel shaped with cricoid plate tilted backwards (maximum constriction at subglottis)	Tubular
c. Situation	High up, below base of tongue with lower end of cricoid at middle of C6 vertebrae	Lower down in neck with lower end of cricoid at lower border of C6 vertebrae
d. Axis of air entry	Straighter	Curved
e. Submucous tissue	Loose, lax, less fibrous	Less so
f. Lymphatics	Richer	Less so
g. Consistency	Softer and pliable	Hard
h. Epiglottis	Large and often foled onto itself	Not so
i. Cough reflex	Weaker	Stronger
j. Neuromuscular mechanism	Unstable	Stable

Significance

- Greatest choke of infantile larynx at subglottic region predisposes them to stridor
- Due to higher position of infantile larynx, axes of pharynx, larynx and trachea are almost in straight line thus air entry in them is straighter whereas in adult life they meet at more acute angle.

6. Pyriform fossa.

- Pyriform fossa is a mucus covered, pear shaped deep depression on lateral wall of laryngopharynx on each side of laryngeal inlet.
- Also called pyriform sinus.

Extent

- From pharygoepiglottic fold to upper end of esophagus.

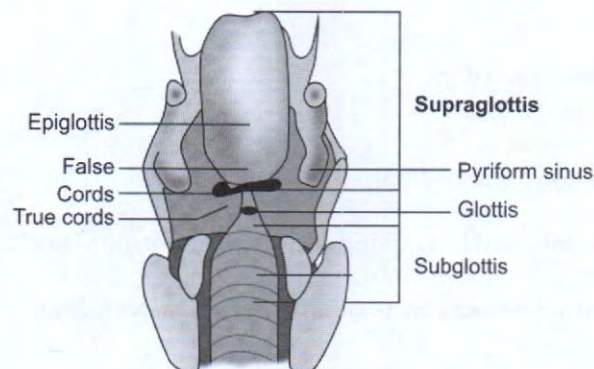


Figure 11: Pyriform fossa

Boundaries

Medially	Laterally	Superiorly
<ul style="list-style-type: none"> ♦ Aryepiglottic fold ♦ Posterolateral surfaces of arytenoid and cricoid cartilages 	<ul style="list-style-type: none"> ♦ Mucous membrane covering medial surface of lamina of thyroid cartilage ♦ Thyrohyoid membrane 	<ul style="list-style-type: none"> ♦ Pharyngoepiglottic fold

Applied Anatomy

- Piriform fossa forms lateral channel for food
- It acts as a catch point for foreign body
- Internal branch of superior laryngeal nerve running submucosally in lateral wall of sinus is accessible for local anesthesia and may also refer pain of carcinoma in piriform fossa to ear
- Sometimes it is artificially deepened to smuggle materials.

7. Herpes Zoster.

- Herpes zoster is a varicella virus causing infection of external ear called herpes zoster oticus.

Herpes Zoster Oticus

- Also called Ramsay Hunt's syndrome or herpes auricularis.

Etiology	Site of affection
<ul style="list-style-type: none"> ♦ Herpes zoster virus 	<ul style="list-style-type: none"> ♦ External ear ♦ Geniculate ganglion of facial nerve ♦ IX and X cranial nerves (sometimes)

Clinical Features

- Common in adults.

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Severe ear pain ♦ Vesicular eruptions on external auditory canal or pinna ♦ Disturbance of hearing and balance (sometimes). 	<ul style="list-style-type: none"> ♦ Vesicular eruptions on concha, antihelix, antitragus or external canal of ear in epidermis and associated with swelling of prickly cells called balloon cells ♦ Facial nerve paralysis (lower motor neuron type) ♦ Facial anesthesia.

Treatment**Conservative**

- Prednisolone (drug of choice)
 - 1 mg/kg/day in 2 divided doses for 5 days.
 Follow up after 5 days to assess progress
 - Paralysis incomplete or recovering
 - Tapering dose for next 5 days (total 10 days)
 - Paralysis complete
 - Continue for next 10 days followed by tapering dose for next five days (total 20 days).
- Acyclovir
 - 800 mg orally or 200 mg IV every 5 hours for 10 days for herpes zoster palsy.
- Antibiotics
 - To prevent secondary infection.
- Analgesics
 - To relieve pain.

8. Anaerobic infection in ENT.

- Anaerobic infection in ENT is not uncommon.

Source of Infection

- Oropharyngeal flora.

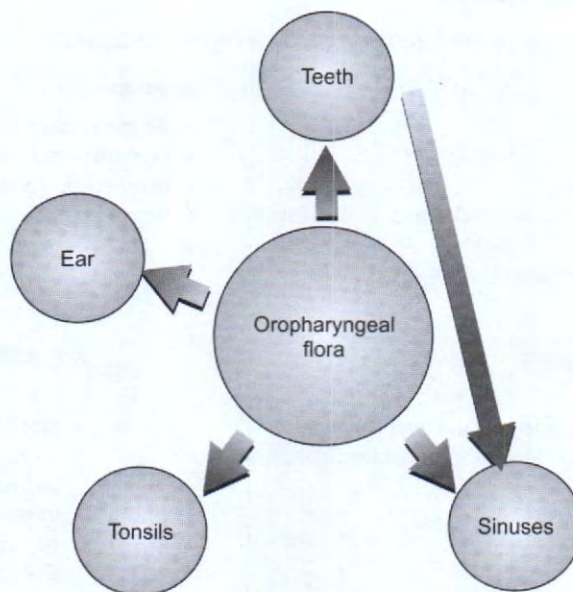


Figure 12: Source of anaerobic infection in ENT

Common Anaerobic Infections and Pathogens

Infection (% of cases)	Anaerobes
a. Acute otitis media (5–15%)	Rarely, <i>Peptostreptococcus</i> species, <i>Propionibacterium acnes</i>
b. Chronic otitis media (50%) and mastoiditis (96%)	<i>Prevotella</i> species, <i>Porphyromonas</i> species, anaerobic cocci
c. Acute rhinosinusitis (5–10%)	<i>Peptostreptococcus</i> species
d. Chronic rhinosinusitis (66%)	<i>Prevotella</i> species, <i>Porphyromonas</i> species, anaerobic cocci
e. Acute tonsillitis	Rarely anaerobes
f. Chronic tonsillitis (75%)	<i>Bacteroides</i> species, <i>Fusobacterium</i> species, <i>Prevotella</i> species, anaerobic cocci.

Investigations

- Culture and sensitivity
 - Investigation of choice
 - Done using pus, blood or secretions from lesions.

Treatment

Specific

- Antimicrobial therapy
 - Directed at the eradication of anaerobic bacteria.

Drug of choice

- Clindamycin
- Metronidazole. (10–21 day)

Duration

- At least 10–21 days (may be extended up to 3 month).

Ref:

1. <http://anaerobicinfections.blogspot.in/p/ent-and-head-neck-infections.html> accessed on 2nd March 2012.

9. Newer generation antihistaminics.

- Newer generation antihistaminics are second generation H₁ receptor antagonists.

Properties	Advantages
<ul style="list-style-type: none"> ♦ Higher H₁ selectivity ♦ Longer duration of action ♦ Absence of anticholinergic side effects ♦ Absence of CNS depressant property or sedation due to poor permeability of Blood-brain-barrier ♦ Additional antiallergic mechanism apart from histamine blockade 	<ul style="list-style-type: none"> ♦ No impairment of psychomotor performance ♦ Produce no subjective effects ♦ No sleepiness or drowsiness ♦ Do not potentiate alcohol or benzodiazepines

Drugs (Dose- Indications)	Pharmacology	Adverse effects
a. Terfenadine (No longer used)	<ul style="list-style-type: none"> ♦ First non-sedative antihistamine ♦ Rapid onset and moderate duration of action 	<ul style="list-style-type: none"> ♦ Blocks cardiac K⁺ channel in overdose and occasionally produces polymorphic ventricular tachycardia ♦ Increased risk of cardiotoxicity in liver diseases or concurrent administration of inhibitors of CYP 3A4 ♦ Cardiotoxicity precipitated by erythromycin, clarithromycin, ketoconazole and itraconazole
b. Fexofenadine (120 mg OD—allergic rhinitis)	<ul style="list-style-type: none"> ♦ Active metabolite of terfenadine devoid of any cardiotoxicity ♦ Rapidly absorbed, has duration of action of 24 hours and excreted unchanged in urine and bile 	<ul style="list-style-type: none"> ♦ Erythromycin and ketoconazole increases its blood levels
c. Astemizole (10 mg OD—for maintenance in perennial rhinitis)	<ul style="list-style-type: none"> ♦ Similar to terfenadine ♦ Slow onset and long duration of action (2–5 days) ♦ Excreted mainly in faeces 	<ul style="list-style-type: none"> ♦ Has potential of producing ventricular tachycardia ♦ Increased appetite, weight gain and flatulence ♦ Adverse effects enhanced by SSRIs, guanidine, some macrolides, azole antifungals and HIV protease inhibitors.
d. Loratidine (10 mg OD)	<ul style="list-style-type: none"> ♦ Long acting selective peripheral H₁ antagonist ♦ Partly metabolized by CYP3A4 ♦ Does not produce cardiac arrhythmia in overdose 	
e. Desloratadine (5 mg OD)	<ul style="list-style-type: none"> ♦ Major active metabolite of loratidine ♦ Inhibits non-histamine mediated release of various inflammatory mediators (additional anti-inflammatory action) 	
f. Cetirizine (10 mg OD—upper respiratory allergies, pollinosis)	<ul style="list-style-type: none"> ♦ Metabolite of hydroxyzine with marked affinity for peripheral H₁ receptors ♦ Not metabolized. ♦ Doesn't show any cardiac toxicity alone or along with erythromycin or ketoconazole ♦ Also inhibits release of histamine and of cytotoxic mediators from platelets as well as eosinophil chemotaxis during secondary phase of allergic response. 	

Contd...

Contd...

Drugs (Dose- Indications)	Pharmacology	Adverse effects
g. Levocetirizine (5–10 mg OD)	♦ Active enantiomer of cetirizine	
h. Azelastine (4 mg OD/0.28 mg Intranasally—seasonal and perennial allergic rhinitis)	♦ Newer H ₁ blocker with good topical activity. ♦ In nasal mucosa acts by down regulating intracellular adhesion molecule-1 expression on nasal mucosa ♦ Also inhibits histamine release and inflammatory reac- tion triggered by LTs and PAF and has bronchodilator property ♦ Metabolism is inhibited by CYP 3A4 inhibitors	♦ Stinging in nose, altered taste perception and weight gain
i. Mizolastine (10 mg OD—allergic rhinitis)	♦ Newer nonsedative antihistaminic	
j. Ebastine (10 mg OD—allergic rhinitis)	♦ Newer nonsedative antihistaminic acting through its active metabolite carbastine	♦ Prolongation of Q-Tc interval (in animal studies)
k. Rupatadine (10 mg OD—allergic rhinitis)	♦ Recently introduced antihistaminic with additional PAF antagonistic property	

Therapeutic uses in ENT

- a. Allergic disorders
 - Antihistaminics though do not suppress the antigen-antibody reaction but is **used as palliatively as they block the effects of released histamine**
 - Particularly useful in certain **immediate type of allergies** like allergic rhinitis (seasonal or perennial)
 - **Type I** hypersensitivity to drugs is suppressed.
- b. Common cold
 - Offers symptomatic relief by **reducing rhinorrhea** (anticholinergic action) and sedation.

10. Otomycosis.

- Otomycosis is fungal infection of external ear canal.

Etiopathogenesis

Causative organisms	Predisposing factors	Pathogenesis
♦ <i>Aspergillus niger</i> (MC) ♦ <i>Aspergillus fumigates</i> ♦ <i>Candida albicans</i>	a. Exposure to contaminated water – Swimming in infected water b. Climatic conditions – Common in hot and humid climate of tropical and subtropic regions – Increased incidence during rainy season c. Topical antibiotics therapy – Favors uninhibited growth of fungus by suppressing local bacterial flora	♦ After entering into external ear, fungus grows luxuriously and uninhibited under favorable conditions of humidity and body heat and suppression of local bacterial flora

Clinical Features

Symptoms	Signs
♦ Intense itching in ear ♦ Discomfort or pain in ear ♦ Brownish or blacking discharge with musty odour ♦ Ear blockage ♦ Hearing loss	♦ Sudden, red and edematous skin of external ear ♦ Fungal mass in ear canal appears like cotton like growth initially but changes to that of wet piece of filter paper with characteristic color – Black headed filamentous growth— <i>Aspergillus niger</i> – Pale blue or green— <i>Aspergillus fumigates</i> – White or creamy— <i>Candida albicans</i>

Investigations

<i>Ear swabs</i>	<i>Blood sugar estimation</i>
♦ For culture and sensitivity and fungal smear	♦ To rule out diabetes in severe and recurrent cases

Treatment*Conservative*

<i>Supportive</i>	<i>Specific</i>
<ul style="list-style-type: none"> ♦ Ear toilet to remove all discharge and epithelial debris conducive for fungal growth either by syringing, suction or mopping ♦ Keeping ear dry ♦ Treatment of associated bacterial infections or diabetes ♦ Analgesics to relieve pain 	<ul style="list-style-type: none"> ♦ Antifungal therapy <p><i>Drugs and dosage</i></p> <ul style="list-style-type: none"> – 1% clotrimazole and tolnaftate (broad spectrum antifungal) – Nystatin in glycerin drops against <i>Candida</i> – Povidine iodine – 2% salicylic acid in alcohol (keratolytic, i.e. removes superficial epidermal layer along with fungal mycelia growing into it) – 1% gentian violet <p><i>Duration</i></p> <ul style="list-style-type: none"> – 1 week even after apparent cure to avoid recurrence

11. Midline granuloma.

- An uncommon condition characterized by apparent chronic inflammatory granulation tissue in nose with rapid destruction of nose and midfacial region and little systemic disturbances
- Also called Stewarts granuloma, lethal midline granuloma, malignant nasal lymphoma or polymorphic reticulosis.

Pathology

- Infiltration by lymphocytes along with plasma cells and lymphoreticular cells (histiocytic lymphoma)
- Necrosis of tissues in midline.

Clinical Features

<i>Symptoms</i>	<i>Signs</i>
♦ Swelling of nose, vestibule and nasal septum initially	♦ Slow progressive destruction and necrosis of cartilage and bone of nose and midfacial structures like palate followed by deformity

Investigations

- Biopsy.

Treatment

<i>Radiotherapy</i>	<i>Operative</i>
♦ Full dose curative radiotherapy to midfacial region and regional lymph nodes	♦ Surgical debridement followed by nasal prosthesis

12. Mucormycosis.

- Mucormycosis is a fungal infections of nose and paranasal sinuses.

Etiology

<i>Causative organism</i>	<i>Predisposing factors</i>
<ul style="list-style-type: none"> ♦ Mucormycosis 	<ul style="list-style-type: none"> ♦ Dry and hot climate ♦ Prolonged use of topical steroids ♦ Immunocompromised individuals ♦ Diabetes ♦ Chronic renal failure ♦ HIV infection ♦ Prolonged systemic steroids ♦ Occupation like farming, garbage cleaning, etc.

Types

<i>Noninvasive form</i>	<i>Invasive form</i>	<i>Fulminant form</i>	<i>Allergic form</i>
Resembles chronic sinusitis	Seen in immunocompromised cases	Due to widespread hematological and intracranial spread	Seen in young adults with history of asthma or polyps

Clinical Features

- Duration varies from months to years.

<i>Symptoms</i>	<i>Signs</i>
<ul style="list-style-type: none"> ♦ Thick purulent nasal discharge, occasionally blood stained ♦ Nasal obstruction ♦ Epistaxis ♦ Swelling of cheek or orbit ♦ Proptosis, diplopia, visual impairment ♦ Severe headache (due to intracranial extension) ♦ Halitosis and cacosmia 	<ul style="list-style-type: none"> ♦ Mass in middle meatus ♦ Thick purulent nasal discharge in middle meatus ♦ Swelling of cheek or orbit or proptosis ♦ Unilateral polyposis

Investigations

<i>Nasal swab</i>	<i>Radiology</i>	<i>Biopsy of nasal mass</i>	<i>Diagnostic nasal endoscopy</i>
<ul style="list-style-type: none"> ♦ For fungal culture 	<ul style="list-style-type: none"> ♦ X-ray or CT shows areas of hyperattenuation due to calcareous deposits ♦ MRI to detect invasive fungal sinusitis 		

Treatment

<i>Conservative</i>	<i>Operative</i>
a. Antifungal drugs (topical/systemic) <ul style="list-style-type: none"> – Amphotericin B for mucormycosis 	<i>Operative</i> <ol style="list-style-type: none"> Functional endoscopic sinus surgery <ul style="list-style-type: none"> – For surgical debridement and establishment of drainage Exenteration and craniofacial resection <ul style="list-style-type: none"> – For fulminant form

Complications

- Orbital cellulitis
- Orbital abscess
- Meningitis
- Brain abscess
- Cavernous sinus thrombosis.

SHORT ANSWERS

13. Laryngeal cartilages.

- Larynx is organ for production of voice and is made up total 9 cartilages, i.e. 3 paired cartilages and 3 unpaired cartilages.

Cartilages of Larynx

Unpaired cartilages	Paired cartilages
♦ Thyroid	♦ Arytenoid
♦ Cricoids	♦ Corniculate
♦ Epiglottic	♦ Cuneiform

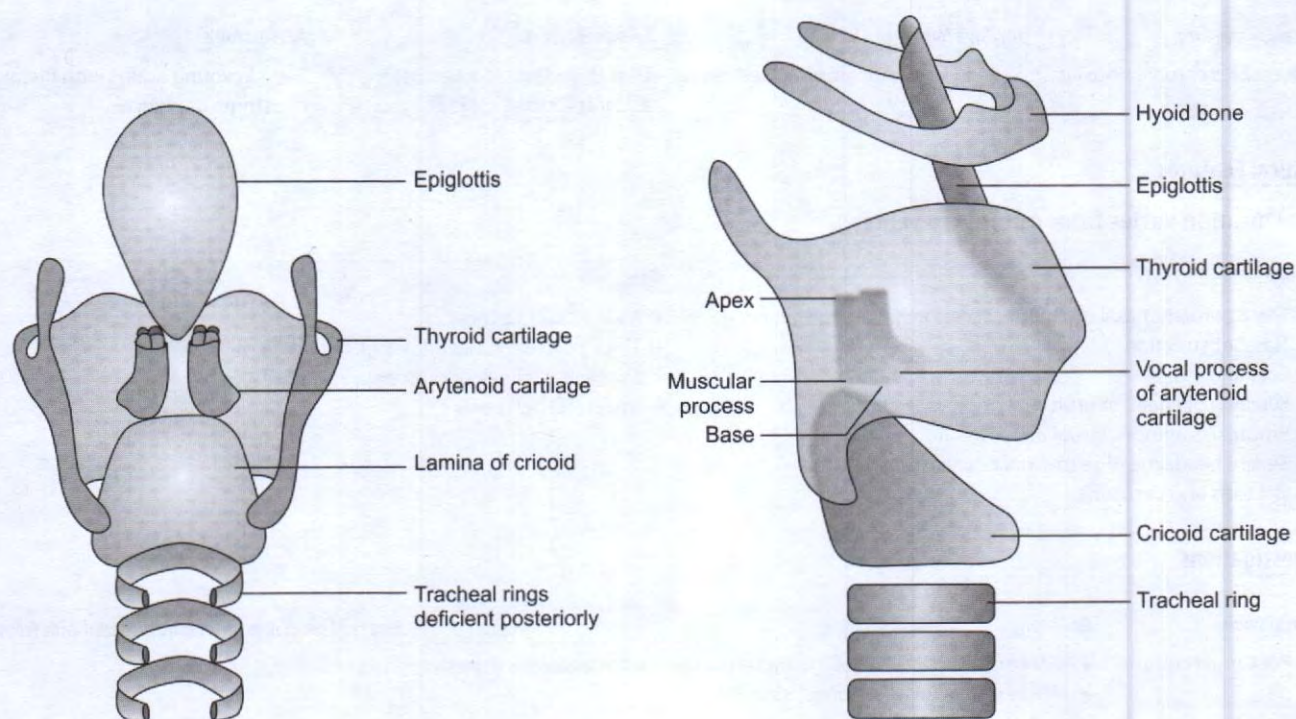
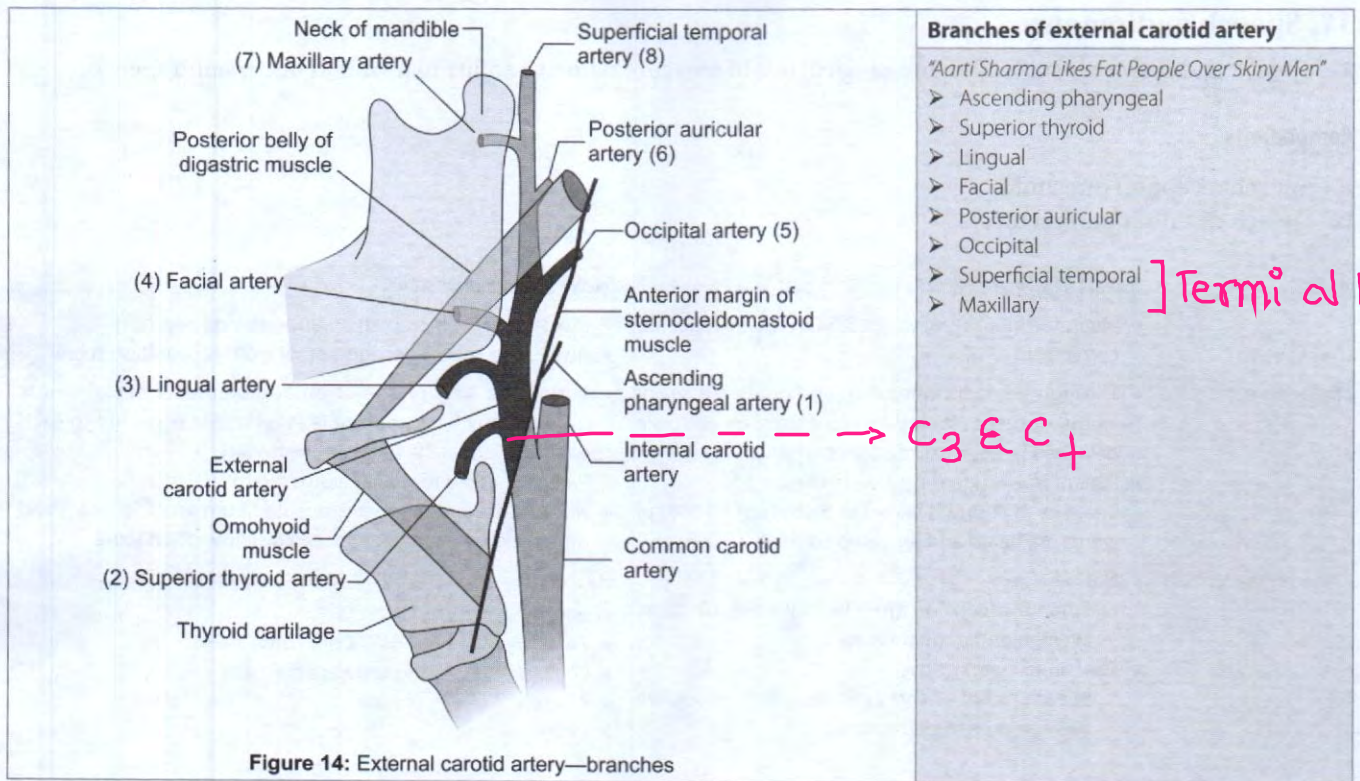


Figure 13: Cartilages of larynx

14. External carotid artery branches.

- External carotid artery is one of terminal branches of common carotid artery which is principle artery to supply structures in front of neck and face.

Origin	Course	Termination
♦ From common carotid artery in carotid triangle at level of upper border of thyroid cartilage opposite intervertebral disc between 3rd and 4th cervical vertebrae	♦ After its origin, it runs upwards and slightly backwards and laterally ♦ Course is slightly curved such that it is anteromedial to internal carotid artery in its lower part, and anterolateral to internal carotid artery in its upper part ♦ It is comparatively superficial in carotid triangle lying under cover of anterior border of sternocleidomastoid	♦ By dividing into maxillary and superficial temporal arteries above carotid triangle, behind neck of mandible



Branches (*Aarti Sharma Likes Fat People Over Skinny Men*)

Medial	Anterior	Posterior	Terminal
a. Ascending pharyngeal artery (1) – Arises from deep aspect of external carotid artery just above its lower end	a. Superior thyroid artery (2) – Arises from front of external carotid just below the level of greater cornu of hyoid bone b. Lingual artery (3) – Arises from front of external carotid artery opposite tip of greater cornu of hyoid bone c. Facial artery (4) – Arises from front of external carotid a little above origin of lingual artery	a. Posterior auricular artery (6) – Arises from back of external carotid just above level at which posterior belly of digastric crosses it b. Occipital artery (5) – Arises from back of external carotid opposite origin of facial artery	a. Superficial temporal artery (8) – Begin behind neck of mandible, in substance of parotid gland b. Maxillary artery (7) – Begin behind neck of mandible, in substance of parotid gland

Significance

- External carotid artery is a principle artery of head and neck region, it knowledge of its anatomy is essential for any otorhinolaryngology surgery.

15. Vestibular schwannoma—clinical features.

Refer Question No. 7 December 2011 (RS2).

16. Hearing aids.

Refer Question No.10 December 2011 (RS2).

17. Speech audiometry.

- Speech audiometry is subjective audiological test to measure patient's ability to hear and understand speech.

Components

- Speech reception threshold.
- Speech discrimination score.

	Speech reception threshold	Speech discrimination score												
	<ul style="list-style-type: none">♦ Minimum intensity at which 50% words are repeated correctly	<ul style="list-style-type: none">♦ Measures patient's ability to understand speech♦ Also called speech recognition or word recognition score												
Procedure	<ul style="list-style-type: none">♦ Through a headphone of an audiometer, a set of words (double syllable with equal stress on both) are delivered to each ear of patient♦ Patient is asked to repeat words heard♦ Intensity of sound is varied in 5 dB steps till 50% of words are heard and repeated correctly	<ul style="list-style-type: none">♦ Through a headphone of an audiometer, a set of words (phonetically balanced single syllable) are delivered to each ear of patient at 30-40 dB above his SRT♦ Patient is asked to repeat words heard♦ Number of words correctly repeated from set of 50 words and multiplied by 2 provides speech discrimination score												
Interpretation	<ul style="list-style-type: none">♦ Normal<ul style="list-style-type: none">– Speech reception threshold within 10 dB of average pure tone threshold♦ Functional hearing loss<ul style="list-style-type: none">– Speech reception threshold more than 10 dB than average pure tone threshold	<table><tr><th>SD score</th><th>Ability to understand speech</th></tr><tr><td>♦ 90–100%</td><td>Normal</td></tr><tr><td>♦ 76–88%</td><td>Slight difficulty</td></tr><tr><td>♦ 60–74%</td><td>Moderate difficulty</td></tr><tr><td>♦ 40–58%</td><td>Poor</td></tr><tr><td>♦ <40%</td><td>Very poor</td></tr></table>	SD score	Ability to understand speech	♦ 90–100%	Normal	♦ 76–88%	Slight difficulty	♦ 60–74%	Moderate difficulty	♦ 40–58%	Poor	♦ <40%	Very poor
SD score	Ability to understand speech													
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♦ 40–58%	Poor													
♦ <40%	Very poor													

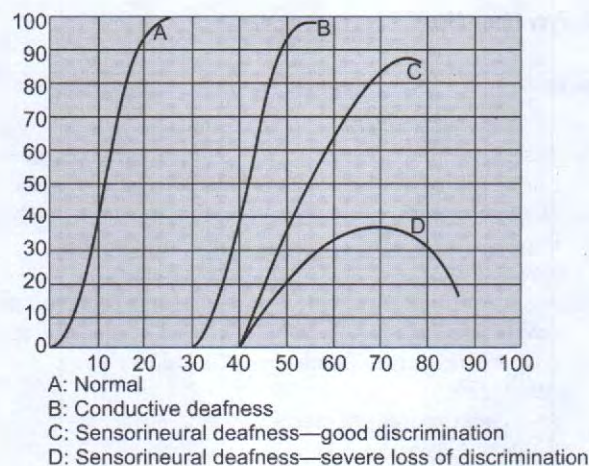


Figure 15: Speech audiometry

Applications

- To find speech recognition threshold.
- To differentiate organic from functional hearing loss.
- To assess PB max
 - PB max is intensity of sound at which patient attains maximum SD score possible which helps to set volume of hearing aid for maximum discrimination.
- To differentiate cochlear and retrocochlear sensorineural hearing loss based on roll over phenomenon
 - Retrocochlear sensorineural hearing loss exhibits roll over phenomenon, i.e. fall in PB word score with increasing speech intensity after reaching a plateau (cochlear sensorineural hearing loss exhibit maintenance of plateau).

Advantages	Disadvantages
<ul style="list-style-type: none"> ♦ Assess actual disability produced by deafness ♦ Predicts usefulness of hearing aid or outcome of surgery ♦ Helps to localize lesion in sensorineural hearing loss 	<ul style="list-style-type: none"> ♦ Subjective test ♦ Need to perform prior pure tone audiometry

18. Vasomotor rhinitis.

Refer Question No. 10 June 2012 (RS2).

19. Rhinitis medicamentosa.

- Rhinitis medicamentosa is an iatrogenic condition characterized by chronic hypertrophic rhinitis.

Etiopathogenesis

Etiology	Pathogenesis
<ul style="list-style-type: none"> ♦ Excessive use of topical decongestant nasal drops more than 1 week (rebound phenomenon) 	<ul style="list-style-type: none"> ♦ Topical nasal decongestant → vasoconstrictor effect → vasoconstriction and ischemia → localized anemia → secondary hyperemia → reflex vasodilatation and rebound phenomenon → increased vascularity with sinusoidal engorgement → nasal obstruction → prompts further use of nasal decongestant ♦ Loss of effect of nasal decongestant due to desensitization of mucosal vessels on prolonged use → irritation of mucosa and dryness

Clinical Features

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Nasal obstruction (due to hypertrophic turbinates) worse at night ♦ Thick viscid nasal discharge ♦ Irritation of nose ♦ Scanty epistaxis ♦ Headache (due to obstruction to sinus ostia) 	<ul style="list-style-type: none"> ♦ Hypertrophied nasal mucosa ♦ Diffuse papillary of anterior end of inferior turbinate and mulberry hypertrophy of posterior end ♦ Crust formation

Treatment

Conservative	Operative
<ul style="list-style-type: none"> ♦ Stop nasal decongestants drops ♦ Short course of local steroids ♦ Oral antihistaminics 	<ul style="list-style-type: none"> ♦ Submucosal diathermy ♦ Surgical reduction of hypertrophied turbinates

20. Complications of mastoidectomy.

- Mastoidectomy is a major operation performed on mastoid air cells to exenteration of disease from mastoid.

Complications

Immediate	Delayed
<ol style="list-style-type: none"> Bleeding <ul style="list-style-type: none"> – Fairly severe, particularly in acute mastoiditis cases Damage to surrounding structures <ol style="list-style-type: none"> Sigmoid sinus <ul style="list-style-type: none"> - Results in severe venous bleeding - Controlled by gelatin sponge kept under pressure of a gauze pack for a minute 	<ol style="list-style-type: none"> Bleeding <ul style="list-style-type: none"> – Reactionary or secondary hemorrhage may occur but rare Effect of damage to surrounding structures <ol style="list-style-type: none"> Sigmoid sinus <ul style="list-style-type: none"> - May result in thrombophlebitis Meninges <ul style="list-style-type: none"> - Extradural abscess, subdural abscess, meningitis or brain abscess may occur

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Immediate	Delayed
ii. Tegmen plate - Results in injury to dura and meninges, causing CSF leakage iii. Facial nerve - May be injured at bend, at vertical portion or horizontal part (in radical mastoidectomy) - Common in children if incision extends below tip of mastoid - Results in facial palsy - Requires immediate decompression iv. Labyrinth (horizontal semicircular canal) - May be injured while clearing mastoid floor or while widening aditus to antrum v. Incus - Dislocated while widening aditus to antrum.	iii. Facial nerve - Facial palsy occurring after 24 hours results due to edema and hematoma - Treated conservatively iv. Labyrinth - Giddiness and sensorineural deafness may develop v. Incus - Conductive deafness due to dislocation of incus c. Infection - Residual infection or recurrence is possible - Infection of external ear pinna may result in perichondritis d. Mastoid fistula - More likely following incision and drainage of mastoid abscess e. Persistence of otorrhea (cavity problem) - Due to inadequate surgery, petrositis, allergy or cholesteatoma f. Stenosis of external auditory canal - If external auditory canal not packed with ribbon gauze properly.

21. Nasal bone fractures.

Refer Question No. 3 December 2010 (RS2).

22. Diagnostic nasal endoscopy.

- Diagnostic nasal endoscopy is examination of nasal cavity using flexible endoscope.

Indications

- Diagnosing diseases of nose or paranasal sinuses
- Locating source of bleeding in epistaxis
- Assessing therapeutic response of nose or paranasal sinuses
- Taking precise biopsy from nose or paranasal sinuses.

Anesthesia

- Topical anesthesia using 4% xylocaine along with a vasoconstrictor (as nasal spray and then by nasal pack).

Position

- Sitting or supine.

Procedure

- Procedure is carried out in three passes.



Figure 16: Diagnostic nasal endoscopy

<i>1st pass</i>	<i>2nd pass</i>	<i>3rd pass</i>
Examination of nasopharynx and inferior meatus	Examination of sphenoethmoidal recess, superior meatus and openings of sphenoid sinus and posterior ethmoidal cells	Detailed examination of middle meatus
<ul style="list-style-type: none"> ♦ Obtain general view of nasal cavity and look for any deviation of septum, septal spurs, any discharge, etc. ♦ Pass endoscope into nasopharynx along floor of nose to examine Eustachian tube openings, nasopharyngeal wall, upper surface of soft palate and uvula ♦ Examine margins of choana, posterior ends of turbinates and openings of nasolacrimal duct in inferior meatus while withdrawing endoscope 	<ul style="list-style-type: none"> ♦ By passing endoscope medial to middle turbinate, examine posterior part of middle turbinate, sphenoethmoidal recess, superior turbinate and meatus, opening of posterior ethmoidal cells and opening of sphenoid sinus 	<ul style="list-style-type: none"> ♦ By passing endoscope from front into middle meatus to examine uncinate process, bulla ethmoidalis, hiatus semilunaris, sinus of turbinate and frontal recess

<i>Advantages</i>	<i>Complications (Disadvantages)</i>
<ul style="list-style-type: none"> ♦ Better visualization ♦ Good illumination ♦ Magnified view 	<ul style="list-style-type: none"> ♦ Hemorrhage ♦ Adhesions ♦ CSF rhinorrhea ♦ Orbital damage, optic nerve

MBBS PHASE III EXAMINATION

DECEMBER 2009

(Revised Scheme 2)

LONG ESSAYS

1. How will you diagnose and manage a case of juvenile nasopharyngeal angiofibroma?

- Juvenile nasopharyngeal angiofibroma is rare but most common benign tumor of nasopharynx.

Etiopathogenesis (Theories of Origin)

<i>Fibroblastic theory</i>	<i>Hormonal theory</i>	<i>Hamartomatous theory</i>	<i>Other theories</i>
Suggests abnormal growth from certain nasopharyngeal tissues like fascia basalis, embryonic occipital cartilage or periosteum of nasopharyngeal wall	Suggest imbalance between estrogen and androgen	Suggest origin from nasal erectile tissue on stimulation by testosterone	Suggest causes, such as trauma, inflammation, infection, allergy and heredity

Site of Origin

- Fibrovascular stroma on posterolateral wall of nasal cavity close to superior margin of sphenopalatine foramen.

Pathology

<i>Gross appearance</i>	<i>Microscopic examination</i>
<ul style="list-style-type: none">♦ Pink or red smooth, firm, unencapsulated mass with lobulated surface	<ul style="list-style-type: none">♦ Tumors show great vascularity with multiple sinusoidal spaces of variable sizes♦ Primitive embryonic vessels lined by flattened epithelium devoid of muscular wall♦ Stroma appears as loose connective tissue, highly cellular with some collagen fibers and fibroblasts

Spread

- Tumor lacks definitive capsule and invades locally
- Earliest spread is medially into nasal cavity and nasopharynx
- Laterally, it extends into pterygopalatine fossa where it erodes posterior wall of maxilla to enter maxillary sinus and then involves infratemporal fossa via pterygomaxillary fissure
- Posteriorly, it may destroy median and lateral pterygoid plates
- Anteriorly, it spreads into orbit and through pterygopalatine fossa into maxillary sinus
- Superiorly, into sphenoidal sinus through sphenopalatine foramen
- Intracranial spread occurs through sphenoidal sinus involving cavernous sinus, sella and middle cranial fossa.

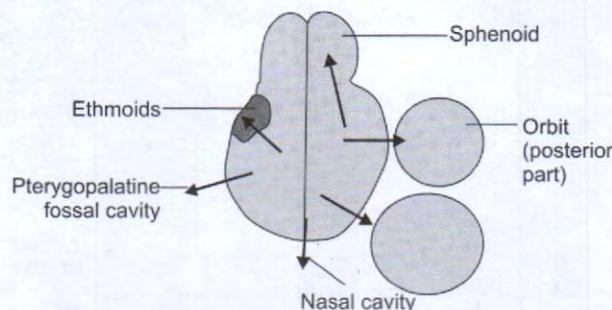


Figure 1: Juvenile nasopharyngeal angiofibroma—spread

Clinical Features

- Exclusively seen in adolescent males in 2nd decade of life.

Symptoms	Signs	
	Early stage	Late stage (due to spread)
<ul style="list-style-type: none"> Progressive increasing blocking of nose Recurrent profuse, painless epistaxis Nasal discharge 	<ul style="list-style-type: none"> Rhinolalia clausa (due to nasal obstruction by tumor) Pink or red smooth globular mass with lobulated surface and dilated blood vessels on surface in posterior part of nasal cavity between middle turbinate and septum on rhinoscopy Nasal septum pushed to opposite side Bulging of soft palate (due to large mass in late stage) Marked anemia (due to repeated blood loss) Underdeveloped secondary sexual characteristics 	<ul style="list-style-type: none"> Middle ear problems like conductive hearing loss, serous otitis media, tinnitus, recurrent otalgia (due to blocking of Eustachian tube) Frog face deformity (proptosis, broadening of nose and swelling of cheek) Eye problems like eye pain, lacrimation, diplopia, etc. Affection of 2nd to 6th cranial nerves (due to involvement of cavernous sinus)

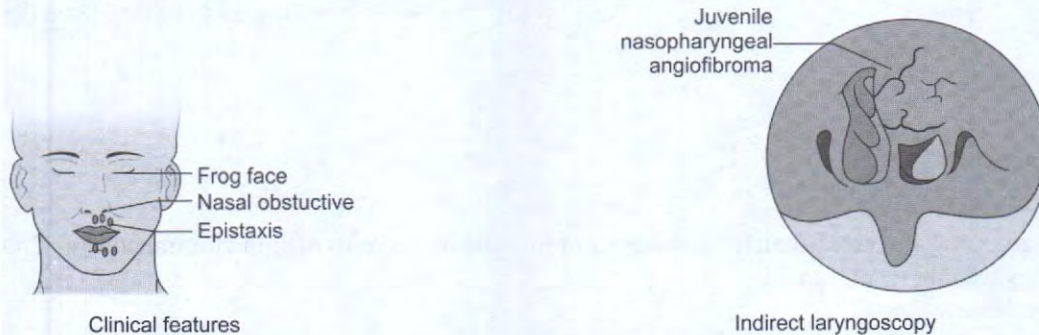


Figure 2: Juvenile nasopharyngeal angiofibroma

Investigations

- Radiography**
 - X-ray shows soft tissue shadow in nasopharynx and forward displacement of posterior wall of maxilla (on lateral view)
 - Water's view shows displacement of nasal septum, opacification of sinuses, widening of distance between maxilla and mandible (if extended into cheek)
 - Anterior bowing of maxilla and posterior bowing of pterygoids (Holman-Miller sign)—Pathognomonic
 - CT scan demonstrates extent of tumor, bony destruction and increased vascularity of tumor
 - MRI useful in intracranial or orbital extensions.
- Angiography**
 - External carotid angiography to demonstrate increased vascularity and feeding vessel (tumor blush).
- Biopsy**
 - Avoided due to risk of severe hemorrhage
 - Excision biopsy preferred.

Staging (Fisch classification)

- Stage I Tumors limited to nasal cavity, nasopharynx with no bony destruction
- Stage II Tumors invading pterygomaxillary fossa, paranasal sinuses with bony destruction
- Stage III Tumors invading infratemporal fossa, orbit and/or parasellar region remaining lateral to cavernous sinus
- Stage IV Tumors invading cavernous sinus, optic chiasmal region and/or pituitary fossa

Differential Diagnosis

<i>Infected antrochoanal polyp</i>	<i>Nasopharyngeal carcinoma</i>
<ul style="list-style-type: none"> ♦ Grey, pale polypoidal mass in nasopharynx ♦ No history of epistaxis 	<ul style="list-style-type: none"> ♦ Friable, proliferative or ulcerated mass ♦ High incidence of lymph node metastases

Treatment*Conservative*

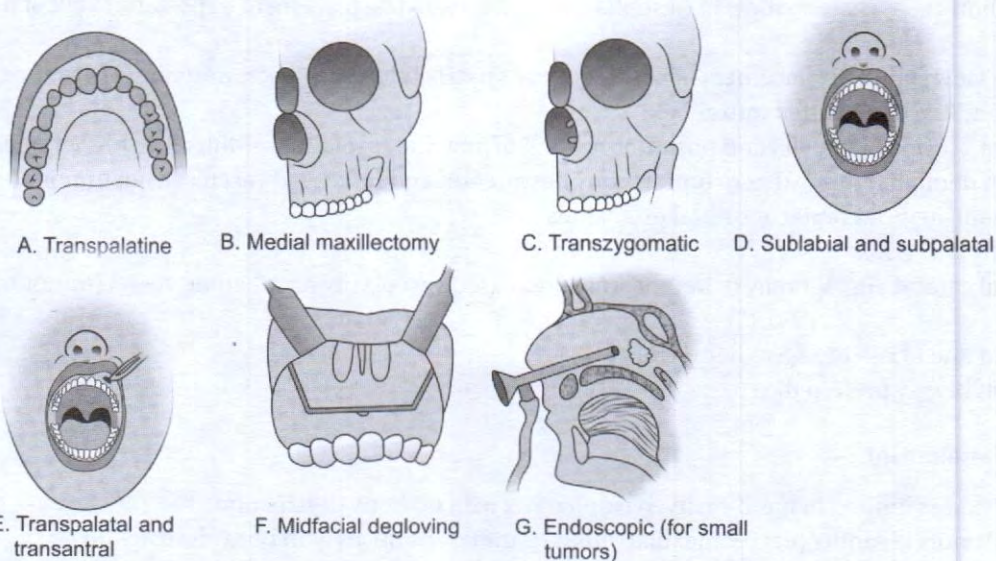
<i>Hormonal therapy</i>	<i>Chemotherapy</i>	<i>Radiotherapy</i>
<ul style="list-style-type: none"> ♦ Doubtful value <i>Indications</i> <ul style="list-style-type: none"> ♦ Patients unfit for surgery ♦ Adjuvant to radiotherapy <i>Drugs</i> <ul style="list-style-type: none"> ♦ Diethylstilboestrol and flutamide 	<i>Indications</i> <ul style="list-style-type: none"> ♦ Recurrent tumors ♦ Residual tumor mass <i>Drugs</i> <ul style="list-style-type: none"> ♦ Doxorubicin, vincristine and dacarbazine 	<ul style="list-style-type: none"> ♦ To reduce vascularity <i>Indications</i> <ul style="list-style-type: none"> ♦ Intracranial extension ♦ Recurrent tumors ♦ Patients unfit for surgery <i>Schedule</i> <ul style="list-style-type: none"> ♦ 3000–3500 cGY in 15-18 fraction in 3–3½ weeks

Operative

- Excision
 - Treatment of choice.

Preoperative preparation

- Blood grouping and cross-matching along with provision of 2–3 pints of blood for transfusion if needed
- Reduce vascularity of tumor by:
 - Cryotherapy
 - Embolization of external carotid artery
 - 2.5 mg Stilboestrol TID for 3 weeks

**Figure 3:** Juvenile nasopharyngeal angiofibroma—surgical approach

Techniques

i. Wilson's transpalatal approach

Indications

- Tumors limited to nasopharynx.

Procedure

- Make a U-shaped incision in coronal plane about 2.5 cm anterior to junction of soft and hard palate
- Remove part of hard palate for better exposure
- Incise mucoperiosteum around tumor mass
- Separate tumor from its base with strong periosteal elevator.

Disadvantages

- Risk of oronasal fistula.

ii. Lateral rhinotomy approach with medial maxillectomy

Indications

- Extension into maxillary sinus.

iii. Transpalatal with Denker's modification of Caldwell-Luc operation

Indications

- Extension into maxillary antrum.

iv. Biller's approach

Indications

- Very large tumor with multiple extensions.

Procedure

- Transmandibular lip splitting to give excellent exposure of infratemporal fossa, pterygoid muscles.

Disadvantages

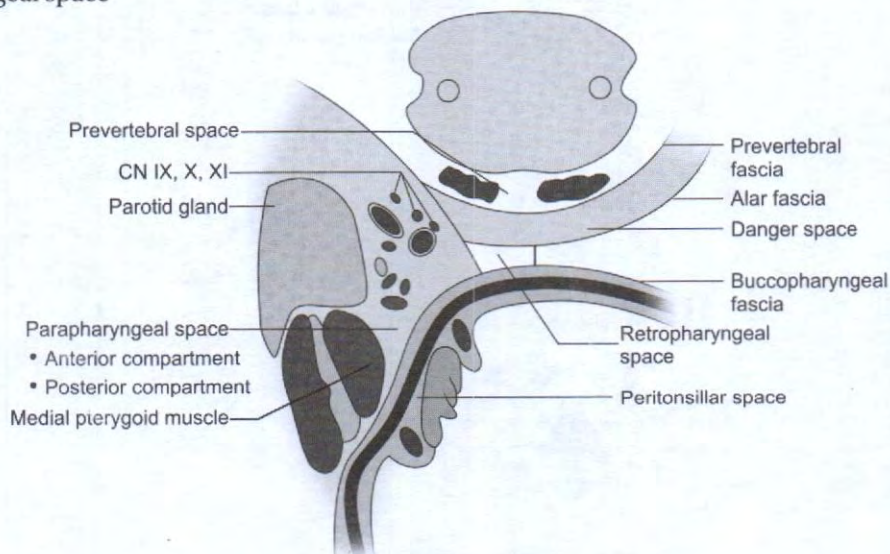
- External scar.

Prognosis

- Good if completely excised.

2. Discuss the etiopathology, clinical features and management of deep neck space infections.

- Deep neck spaces are potential spaces between layers of cervical fascia.
- Of many deep neck spaces, spaces of clinical importance are:
 - i. Retropharyngeal space
 - ii. Parapharyngeal space

Anatomy**Figure 4: Deep spaces of neck—anatomy**

	Retropharyngeal space	Parapharyngeal space
Synonyms	Posterior visceral space Retrosophageal space Retrovisceral space Space of Gillette Lincoln's highway (for spread of infection to mediastinum)	Pharyngomaxillary space Lateral pharyngeal space
Boundaries		
Anterior	Buccopharyngeal fascia	Interpteryoid fascia Pterygomandibular raphe
Posterior	Alar fascia	Prevertebral fascia Posterior aspect of carotid sheath
Lateral	Carotid sheath Parapharyngeal space	Superficial layer of deep cervical fascia Medial pterygoid muscle Mandible Deep surface of parotid gland
Medial	Midline septum (dividing it in to 2 spaces)	Middle layer of deep cervical fascia Fascia of tensor and levator muscles of velum palatine Styloglossus
Superior	Base of skull	Base of skull
Inferior	Extends into superior mediastinum	Hyoid bone
Contents	Loose areolar tissue and fat Lymph nodes draining adjacent structures	<ul style="list-style-type: none"> ♦ Prestyloid compartment Loose areolar tissue and fat Lymph nodes Internal maxillary artery ♦ Poststyloid compartment Carotid artery Jugular vein Cranial nerves IX, X, XI and XII Sympathetic trunk

Deep Neck Space Infections

A. Retropharyngeal abscess

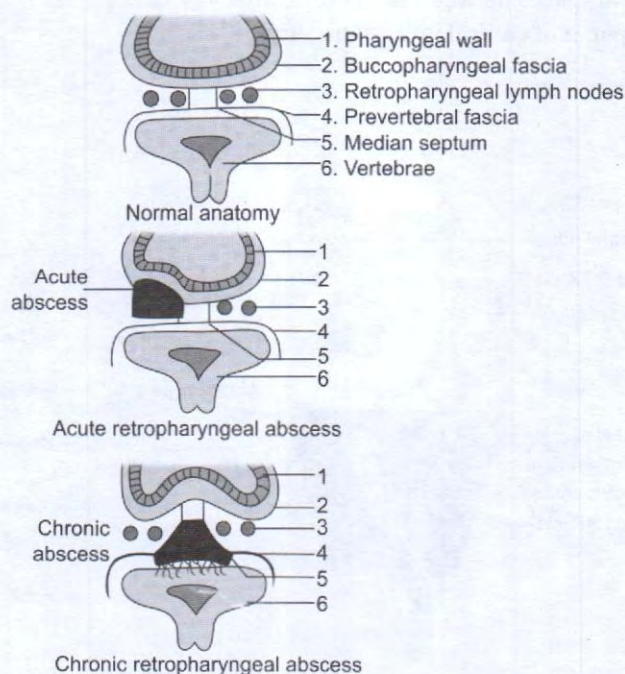


Figure 5: Retropharyngeal abscess

	Acute retropharyngeal abscess	Chronic retropharyngeal abscess
	<ul style="list-style-type: none"> ♦ Very painful condition due to suppuration of retropharyngeal lymph nodes 	<ul style="list-style-type: none"> ♦ Chronic tuberculous infection of retropharyngeal lymph nodes <p>Types</p> <ol style="list-style-type: none"> Lateral type <ul style="list-style-type: none"> – Due to tuberculous infection of retropharyngeal lymph nodes – Presents unilateral swelling on right or left side of midline – Usually affects children aged 8–10 years Central type (more common) <ul style="list-style-type: none"> – Due to cold abscess of cervical spine – Presents midline swelling spreading to both sides – Affects any age group
Etiology		
Causative organism	<ul style="list-style-type: none"> ♦ <i>Streptococcus</i> ♦ <i>Staphylococcus</i> 	<ul style="list-style-type: none"> ♦ <i>Mycobacterium tuberculosis</i>
Source of infection	<ul style="list-style-type: none"> ♦ Secondary to infections of adenoids, nasopharynx, sinuses, nasal cavity (in children) ♦ Penetrating injury to posterior pharyngeal wall or cervical esophagus (in adults) ♦ Spread of pus from acute mastoiditis 	<ul style="list-style-type: none"> ♦ Cold abscess of cervical spine ♦ Tuberculous infection of retropharyngeal lymph nodes secondary to tuberculosis of deep cervical nodes
Clinical features	<ul style="list-style-type: none"> ♦ Commonly seen in boys below 3 years of age 	<ul style="list-style-type: none"> ♦ Insidious onset ♦ Systemic features of tuberculosis
Symptoms	<ul style="list-style-type: none"> ♦ Difficulty in breathing and eating (due to obstruction of air and food passage by abscess) ♦ Croupy cough ♦ Voice like quacking of a duck ♦ Constitutional symptoms like fever, toxemia 	<ul style="list-style-type: none"> ♦ Discomfort in throat ♦ Difficulty in swallowing (not marked) ♦ Painless lump in throat
Signs	<ul style="list-style-type: none"> ♦ Unilateral swelling in posterior pharyngeal wall (on one side of midline) ♦ Soft, fluctuating swelling extending upwards toward nasopharynx and downwards into cricopharynx but never crossing midline ♦ Glossy inflamed mucosa ♦ Dribbling and pooling of saliva ♦ Torticollis ♦ Trismus ♦ Stridor ♦ Acute lymphadenitis of jugulodigestic lymph nodes or lymph nodes in upper part of posterior triangle 	<ul style="list-style-type: none"> ♦ Fluctuant swelling on posterior pharyngeal wall located centrally (due to cervical spine caries) or one side of midline (due to tuberculous lymph nodes) ♦ No signs of acute inflammation ♦ Torticollis
Investigations	<ol style="list-style-type: none"> Radiology (X-ray, CT and MRI) <ul style="list-style-type: none"> – Lateral view of neck shows widening of prevertebral shadow and presence of fluid level and gas – Increased distance between laryngotracheal air column and anterior border of cervical vertebra (at C2 >7 mm and at C6 >14 mm in children and >22 mm in adults) 	<ol style="list-style-type: none"> X-rays <ul style="list-style-type: none"> – Lateral view reveals caries of spine and soft tissue shadow of abscess Tuberculin test <ul style="list-style-type: none"> – Positive Bacteriological examination of pus <ul style="list-style-type: none"> – Reveals mycobacteria
Differential diagnosis	<ul style="list-style-type: none"> ♦ Cold abscess of retropharyngeal lymph nodes ♦ Cold abscess of vertebral column 	

Contd...

	<i>Acute retropharyngeal abscess</i>	<i>Chronic retropharyngeal abscess</i>
Treatment		
Supportive	a. Tracheostomy – In cases of large abscess causing mechanical obstruction to airway or laryngeal edema	
Conservative	♦ Systemic antibiotics to control infection ♦ Analgesics to relieve pain and control inflammation ♦ Steroids for impending laryngeal edema ♦ Nutrition	♦ Antitubercular treatment (Full course as per RNTCP)
Operative	a. Incision and drainage Anesthesia – Nil Position – Supine with head low Incision – Vertical incision in most fluctuant area of abscess Procedure – Open mouth with gag and incise abscess – Drain abscess with suction (prevent aspiration of pus) Postoperative – Suitable antibiotic cover for 7–10 days	a. Incision and drainage – Vertical incision along border of sternomastoid (anterior border for low abscess or posterior border for high abscess) and drainage with suction b. Aspiration – Aspiration of abscess with thick needle through oral cavity under cover of antibiotics and antitubercular drugs
Complications	♦ Laryngeal edema requiring tracheostomy ♦ Dehydration due to diminished fluid intake ♦ Spontaneous rupture leading to sudden death due to inhalation of pus	

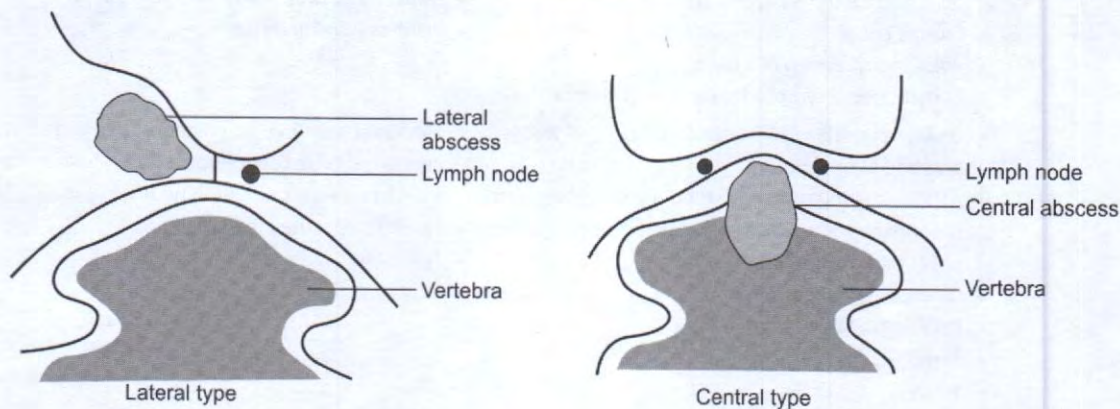


Figure 6: Chronic retropharyngeal abscess—types

B. Parapharyngeal abscess

- Suppuration of parapharyngeal space due to spread of infection from adjacent structures.

Etiology

<i>Spread of infection from</i>	<i>External trauma</i>
<ul style="list-style-type: none"> ♦ Pharynx—Acute and chronic infections of tonsils and adenoids, bursting of peritonsillar abscess ♦ Teeth—Infection of lower last molar teeth ♦ Ear—Bezold's abscess, petrositis ♦ Other spaces—Parotid, retropharyngeal and submaxillary space infections 	<ul style="list-style-type: none"> ♦ Penetrating injury to neck ♦ Injection of local anesthetics for tonsillectomy or mandibular nerve block

Clinical features

- Common in young adults.

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Fever ♦ Painful swallowing ♦ Sore throat and pain 	<ul style="list-style-type: none"> ♦ Torticollis ♦ Toxemia <p><i>Anterior compartment infections</i></p> <ul style="list-style-type: none"> ♦ Prolapse of tonsil and tonsillar fossa ♦ Trismus (due to spasm of medial pterygoid muscle) ♦ Swelling behind angle of jaw <p><i>Posterior compartment infections</i></p> <ul style="list-style-type: none"> ♦ Swelling of pharynx behind posterior pillar ♦ Paralysis of IX, X, XI, XII cranial nerves and sympathetic chain ♦ Swelling of parotid region

Differential diagnosis

- Peritonsillar abscess (quinsy)
- Retropharyngeal abscess
- Tumors
- Aneurysms.

Treatment

Conservative	Operative
<ul style="list-style-type: none"> ♦ Systemic antibiotics intravenously to control infection ♦ Analgesics to relieve pain and control inflammation 	<p>a. Incision and drainage</p> <p><i>Anesthesia</i></p> <ul style="list-style-type: none"> – General <p><i>Incision</i></p> <ul style="list-style-type: none"> – Horizontal incision, 2–3 cm below angle of mandible <p><i>Procedure</i></p> <ul style="list-style-type: none"> – After incision, carry out blunt dissection along inner surface of medial pterygoid towards styloid process – Evacuate abscess and retain a drain

Complications

- Acute edema of larynx with respiratory obstruction
- Thrombophlebitis of jugular vein with septicemia
- Spread of infection to retropharyngeal space
- Spread of infection to mediastinum along carotid space
- Mycotic aneurysm of carotid artery (due to weakening of vessel wall)
- Carotid blow out with massive hemorrhage.

SHORT ESSAYS**3. Intratemporal complications of CSOM (Chronic suppurative otitis media).**

- Extension of infection middle ear (suppurative otitis media) to adjacent structures is common if not properly treated.

Factors Influencing Development of Complications

a. Age	Common in children and elderly
b. Poor socioeconomic condition	Poor health education, lack of personal hygiene, limited access to health care predispose to complications
c. Virulence of organism	Complications common with virulent organism

d. Improper treatment	Insufficient dose, less effective drug or insufficient duration of treatment can cause complications
e. Immune status	Decreased immunity due to AIDS, uncontrolled diabetes, etc. predisposes to complications
f. Preformed pathways	Preformed pathways due to pathology or ear surgery act as portal of spread of complication
g. Cholesteatoma	Destroys bone and help in deeper penetration of infection

Warning Signs of Impending Complication

- Persistent pain and headache on affected side
- High fever with chills and rigor
- Giddiness (persistent or recurrent)
- Twitching of facial muscles on affected side
- Positive fistula test.

Pathways of Spread of Infection

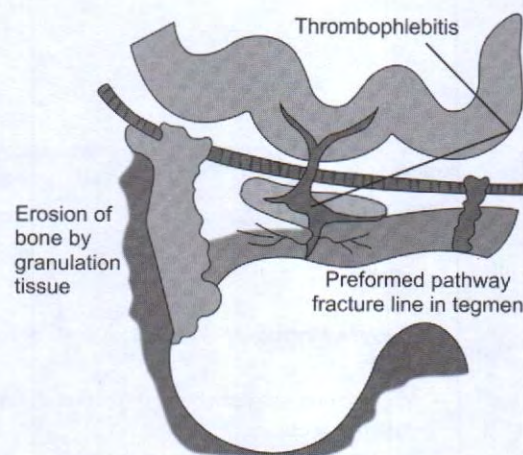


Figure 7: Pathways of spread of middle ear infection

Direct erosion of bone	Retrograde thrombophlebitis	Preformed pathways
<ul style="list-style-type: none"> ♦ Due to hyperemic decalcification or osteitis in acute infections ♦ Due to cholesteatoma or granulation tissue in chronic infections 	<p>(Common in acute infections)</p> <p>Infection of mastoid bone is transmitted through</p> <p>↓</p> <p>Veins of Haversian canals</p> <p>↓</p> <p>Dural veins</p> <p>↓</p> <p>Dural venous sinuses</p> <p>↓</p> <p>Thrombophlebitis of venous sinuses and cortical vein thrombosis</p>	<ul style="list-style-type: none"> ♦ Along anatomical continuity <ul style="list-style-type: none"> – Example: Mastoiditis usually follows otitis media due to anatomical continuity of mastoid air cells with middle ear – Labyrinthitis travels along internal acoustic meatus, aqueduct of vestibule and cochlea to meninges ♦ Congenital dehiscence <ul style="list-style-type: none"> – Example: In bony facial canal, floor of middle ear over jugular bulb ♦ Patent sutures <ul style="list-style-type: none"> – Example: Petrosquamous suture ♦ Surgical defects <ul style="list-style-type: none"> – Example: Stapedectomy, fenestration and mastoidectomy with exposure of dura ♦ Recent or previous skull fractures as skull fractures heal only by fibrous scar ♦ Oval and round windows ♦ Foramina for blood vessels

Spreads Through Different Walls of Middle Ear

Through posterior wall	Through medial wall	Through roof	Through floor
<ul style="list-style-type: none"> ♦ Acute mastoiditis in cellular mastoid ♦ Facial nerve palsy in sclerotic or acellular mastoid ♦ Lateral sinus thrombosis ♦ Cerebellar abscess (Trautman's triangle) ♦ Superior petrosal sinus thrombosis (Solid angle) 	<ul style="list-style-type: none"> ♦ Labyrinthitis 	<ul style="list-style-type: none"> ♦ Extradural abscess ♦ Subdural abscess ♦ Temporal and cerebellar abscess ♦ Meningitis ♦ Brain abscess 	<ul style="list-style-type: none"> ♦ Thrombosis of jugular bulb

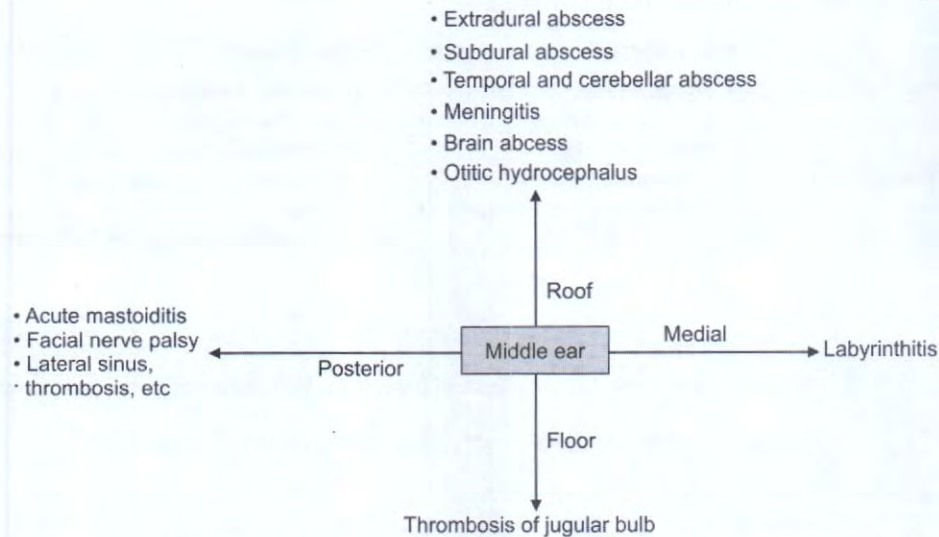


Figure 8: Complications of middle ear infection

Classification

Extracranial		Intracranial
Intratemporal	Extratemporal (Cervical)	
<ul style="list-style-type: none"> ♦ Mastoiditis <ul style="list-style-type: none"> – Acute coalescent* – Masked or latent* – Chronic mastoiditis# ♦ Petrositis ♦ Facial paralysis ♦ Labyrinthitis# 	<ul style="list-style-type: none"> ♦ Subperiosteal abscess* ♦ Zygomatic abscess* ♦ Bezold abscess* ♦ Luc's abscess* ♦ Citelli's abscess* ♦ Thrombophlebitis* of jugular or subclavian vein 	<ul style="list-style-type: none"> ♦ Extradural abscess ♦ Subdural abscess ♦ Meningitis ♦ Brain abscess ♦ Lateral sinus thrombophlebitis ♦ Otitic hydrocephalus

(Note: *Complications of ASOM, #Complications of CSOM, Rest are for both)

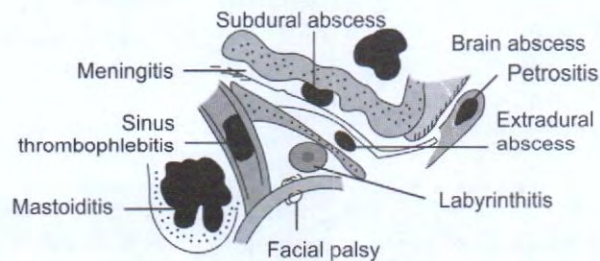


Figure 9: Complications of otitis media

Intratemporal Complications

A. Mastoiditis

- Mastoiditis is involvement of bony walls of mastoid air cells due to spread of middle ear infection from mucosal lining of antrum and mastoid air cells.

Acute mastoiditis

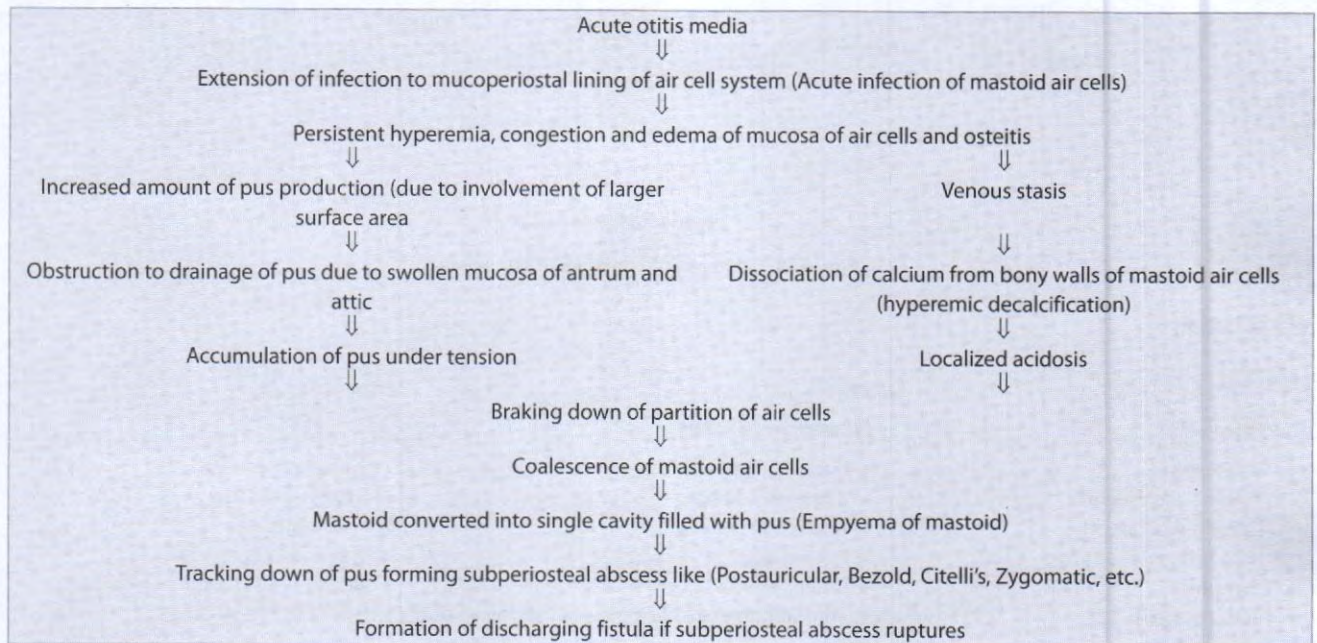
- Acute mastoiditis is coalescence of mastoid air cells due to destruction of bony partition between mastoid air cells
- Also called coalescent mastoiditis or acute surgical mastoiditis

Etiology

- Acute suppurative otitis media—as a complication.

Causative organisms	Mode of infection	Predisposing factors
<ul style="list-style-type: none"> Beta-hemolytic streptococci (MC) Staphylococci Pneumococci Hemophilus influenza Pseudomonas 	<ul style="list-style-type: none"> Spread of infection from middle ear cavity through aditus to mastoid air cells Blood borne infection Trauma like penetrating injuries 	<ul style="list-style-type: none"> Inadequate drainage of exudate Highly virulent organisms Lowered host immunity due to measles, exanthematous fevers, poor nutrition and associated systemic diseases like diabetes Inadequate or improper antibiotic therapy

Pathogenesis



Pathology

Gross appearance	Microscopic examination
<ul style="list-style-type: none"> Smooth-lined large cavity filled with pus Pale bluish epithelium 	<ul style="list-style-type: none"> Pale bluish squamous epithelium Submucous fibrosis

Clinical features

- Commonly affects children.

Symptoms	
a. Pain and swelling behind ear	<ul style="list-style-type: none"> Persistence of pain or increased intensity of pain or recurrence of pain after initial subsiding
b. Fever	<ul style="list-style-type: none"> Persistence or recurrence of fever inspite of adequate antibiotic treatment Fever is often low grade but in children is often high with raised pulse rate

Contd...

Contd...

Symptoms	
c. Ear discharge (persists beyond 3 weeks)	<ul style="list-style-type: none"> ♦ Mucopurulent ear discharge which becomes more profuse and purulent ♦ Sometimes, ear discharge may cease (due to obstruction)
d. Hearing loss	<ul style="list-style-type: none"> ♦ Usually, conductive deafness
Signs	
a. Mastoid tenderness (diagnostic)	<ul style="list-style-type: none"> ♦ Pressure over middle or tip of mastoid process and root of zygoma elicits tenderness ♦ Compare with healthy side
b. Mastoid swelling	<ul style="list-style-type: none"> ♦ Initially, smooth ironed out feel of mastoid due to edema and thickening of periosteum of posterior auricular region ♦ Later, obliteration of retroauricular sulcus with downward and forward pushing of pinna ♦ Finally, superosteal fluctuating, tender and warm abscess is formed once pus bursts through bony cortex
c. Ear discharge	<ul style="list-style-type: none"> ♦ Mucopurulent or purulent discharge, often pulsatile (light house effect) ♦ Cleaning of pus from deep external auditory meatus results in immediate filling of meatus (mastoid reservoir sign)
d. Sagging of meatal wall	<ul style="list-style-type: none"> ♦ Posterosuperior meatal wall sags due to periosteitis of bony wall between antrum and deeper posterosuperior part of bony canal
e. Tympanic membrane	<ul style="list-style-type: none"> ♦ Tympanic membrane usually shows small perforation in pars tensa (central or marginal) with rest of membrane congested ♦ Sometimes, tympanic membrane may be intact but appears dull and opaque (in those who received inadequate antibiotics)
f. Hearing loss	<ul style="list-style-type: none"> ♦ Conductive type of hearing loss
g. Constitutional symptoms	<ul style="list-style-type: none"> ♦ Ill and toxic appearance of patient with fever

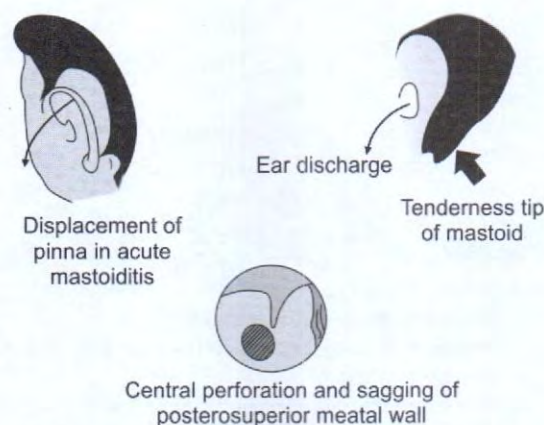


Figure 10: Clinical features of coalescent mastoiditis

Investigations

Tuning fork tests	Blood investigations	Microbiological	Radiological
a. Rinne's test – Negative b. Weber's test – Lateralized to diseased side c. ABC test – Normal	a. Blood count – Polymorphonuclear leucocytosis b. ESR – Usually raised	a. Culture and sensitivity – From ear swab	a. X-ray mastoid – Clouding of air cells (due to collection of exudate) – Indistinct bony partitions between air cells – Cavity formation in later stages

Differentiation diagnosis

<i>Postauricular lymphadenitis</i>	<i>Furunculosis of meatus</i>	<i>Infected sebaceous cyst</i>
<ul style="list-style-type: none"> ♦ No history of preceding otitis media, ear discharge or deafness ♦ Superficial abscess 	<ul style="list-style-type: none"> ♦ No history of preceding otitis media ♦ Painful movements of pinna and extreme tenderness when pressed over tragus or below cartilaginous part of meatus ♦ Confinement of meatal swelling to cartilaginous part only ♦ Purulent discharge (never mucoid or mucopurulent) ♦ Enlargement of pre or postauricular lymph nodes ♦ Mild conductive deafness (due to occlusion of meatus) ♦ Normal tympanic membrane ♦ Clear air cells visualized on X-ray mastoid 	<ul style="list-style-type: none"> ♦ No history of preceding otitis media

Complications

- Abscess formation (abscess in relation to mastoid infection)
 - Postauricular abscess (subperiosteal abscess)
 - Zygomatic abscess
 - Bezold abscess
 - Meatal abscess (Luc's abscess)
 - Behind mastoid (Citelli's abscess)
 - Parapharyngeal or retropharyngeal abscess
- Labyrinthitis
- Facial paralysis
- Petrositis
- Intracranial abscess (extradural or subdural)
- Meningitis
- Brain abscess
- Lateral sinus thrombophlebitis
- Otitic hydrocephalus.

Treatment

Objectives

- Resolution of infection
- Prevention of intracranial infection.

<i>Supportive</i>	<i>Specific</i>	
	<i>Conservative</i>	<i>Operative</i>
<ul style="list-style-type: none"> ♦ Hospitalize patient immediately ♦ NSAIDs to relieve pain ♦ Hot fermentation over affected part to soothen pain 	<ol style="list-style-type: none"> Antibiotic therapy <ul style="list-style-type: none"> – Start with amoxicillin or ampicillin – Change over to specific antibiotics according to culture sensitivity report – Add metronidazole if anaerobic organism suspected – Antibiotic therapy is also continued after operative procedures like mastoidectomy 	<ol style="list-style-type: none"> Myringotomy <ul style="list-style-type: none"> <i>Indications</i> <ul style="list-style-type: none"> – Pus under tension – No response to conservative therapy Mastoidectomy (described below)

Mastoidectomy

- Mastoidectomy is removal of mastoid air cells done for any acute or chronic infection of mastoid air cells or any complication of chronic otitis media involving mastoid.

<i>Indications</i>	<i>Objectives</i>
<ul style="list-style-type: none"> ♦ Subperiosteal abscess ♦ Sagging of posterosuperior meatal wall ♦ Positive reservoir sign ♦ No response to conservative therapy in 48 hours ♦ Impending complications 	<ul style="list-style-type: none"> ♦ Externate all mastoid air cells and remove any pockets of pus

Types

<i>Simple mastoidectomy (Cortical or conservative mastoidectomy/Schwartz operation)</i>	<i>Modified radical mastoidectomy</i>
<ul style="list-style-type: none"> ♦ Aims to drain mastoid only ♦ Indicated if disease limited to mastoid region only ♦ Consists of extenteration of all accessible mastoid air cells preserving posterior meatal wall 	<ul style="list-style-type: none"> ♦ Indicated when disease extends to middle ear cavity ♦ Consists of extenteration of disease from attic and mastoid by removal of posterior meatal and lateral wall and converting mastoid, attic and external ear into single cavity ♦ Tympanic membrane remnant, functioning ossicles and reversible mucosa and function of Eustachian tube are preserved ♦ Does not affect hearing

Masked mastoiditis

- A type of mastoiditis characterized by slow progressive destruction of mastoid air cells without signs and symptoms of acute mastoiditis.

Etiology

- Inadequate antibiotic treatment of acute mastoiditis or otitis media in terms of dose, frequency or duration
- Commonly seen in oral penicillins.

Pathology

- Extensive destruction of air cells with granulation tissue and dark gelatinous material filling mastoid.

Clinical features

- Common in children

<i>Symptoms</i>	<i>Signs</i>
<ul style="list-style-type: none"> ♦ Mild pain behind ear ♦ Persistent hearing loss ♦ No fever or discharge 	<ul style="list-style-type: none"> ♦ Thickened tympanic membrane with loss of translucency ♦ Slight tenderness over mastoid ♦ Absence of mastoid swelling

Investigations

<i>Audiometry</i>	<i>X-ray mastoid</i>
<ul style="list-style-type: none"> ♦ Conductive hearing loss of variable degree 	<ul style="list-style-type: none"> ♦ Clouding of air cells with loss of cell outline

*Treatment**Specific*

- Cortical mastoidectomy followed by full dose antibiotic therapy.

Complications

- Extradural or perisinus abscess due to erosion of tegmen tympani and sinus plate.

B. Petrositis

- Petrositis is the inflammation of petrous part of temporal bone leading to coalescence of petrous apex air cells with formation of empyema
- Manifests as Gradenigo syndrome.

Gradenigo syndrome

- Gradenigo syndrome is triad of (3D's)
 - Diplopia (due to external rectus palsy because of 6th cranial nerve palsy)
 - Deep-seated ear or retro-orbital pain (Trigeminal neuralgia)
 - Discharge from ear—persistent.
- Seen as clinical presentation of petrositis (inflammation of petrous part of temporal bone leading to coalescence of petrous apex air cells with formation of empyema).

<i>Etiology</i>	<i>Spread of infection</i>
<ul style="list-style-type: none"> ♦ Otitis media (acute or chronic) ♦ Mastoiditis (acute, latent, coalescent) 	<ul style="list-style-type: none"> ♦ Posterosuperior tract which starts in mastoid and runs behind or above bony labyrinth to petrous apex ♦ Anteroinferior tract which starts at hypotympanum near Eustachian tube, runs around cochlea to reach petrous apex

Clinical features

- May be acute or chronic in onset.

<i>Symptoms</i>	<i>Signs</i>
<ul style="list-style-type: none"> ♦ Persistent foul smelling ear discharge ♦ Deep-seated ear or retrobulbar pain ♦ Diplopia (due to paralysis of external rectus) ♦ Fever and headache associated with vomiting and neck rigidity ♦ Recurrent vertigo (due to involvement of facial and statoacoustic nerves) 	<ul style="list-style-type: none"> ♦ Facial paralysis

Investigations

- Radiography
 - Plain X-ray (Towne's view and Stenver's view)
 - CT scan of temporal bone shows bony details of petrous apex and air cells
 - MRI helps to differentiate diploic marrow containing apex from fluid or pus.

Treatment

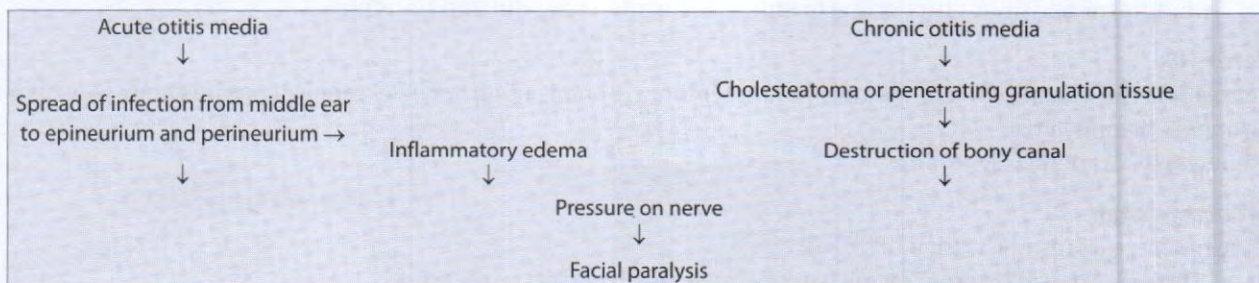
<i>Conservative</i>	<i>Operative</i>
a. Antibiotics <ul style="list-style-type: none"> – High dose intravenous antibiotics preceding and following surgery for 4–5 days after disappearance of symptoms – Usually used is chloramphenicol with flucloxacillin 	<ul style="list-style-type: none"> ♦ Indicated in chronic cases <ul style="list-style-type: none"> a. Mastoidectomy <ul style="list-style-type: none"> - Simple, cortical, modified radical or radical if not already done b. Curettage and drainage <ul style="list-style-type: none"> - Curettage and drainage of fistulous tract to provide free drainage

C. Facial paralysis

- Facial paralysis occurs as complication of both acute and chronic otitis media

Etiology

- Otitis media (acute and chronic)—infranuclear palsy
- Temporal abscess—supranuclear palsy.

Pathogenesis*Clinical features*

- Insidious during onset but slowly progressive.

Treatment

Conservative	Operative
a. Antibiotics <ul style="list-style-type: none"> – To control infection in acute otitis media – Facial paralysis resolves spontaneously if infection 	a. Myringotomy or cortical mastoidectomy <ul style="list-style-type: none"> – To relieve pressure on nerve in acute otitis media if duration of palsy is <24 hours b. Exploration and curettage <ul style="list-style-type: none"> – Urgent exploration of middle ear and mastoid and inspection of facial canal (from geniculate ganglion to stylomastoid foramen) for any encroachment by cholesteatoma or granulation tissue – Any granulation tissue surrounding nerve is removed but if it invades nerve, it is left in place c. Facial nerve decompression <ul style="list-style-type: none"> – Done if ipsilateral facial nerve palsy does not recover in 6–8 weeks after mastoidectomy d. Resection and grafting of nerve <ul style="list-style-type: none"> – If a part of nerve is destroyed by granulation tissue, affected nerve is resected and grafting done following control of infection

D. Labyrinthitis

- Labyrinthitis is the inflammation of labyrinth.

Etiology	Precipitating factors	Spread of infection
<ul style="list-style-type: none"> ♦ Otitis media ♦ Meningitis 	<ul style="list-style-type: none"> ♦ Trauma like fracture of skull, surgical trauma like mastoidectomy or stapedectomy 	<ul style="list-style-type: none"> ♦ Through round or oval window (otitis media) ♦ Through retrograde infection to membranous labyrinth (meningitis) ♦ Along fluid pathway connecting subarachnoid space and perilymphatic space of cochlea (meningitis)

Classification (stages)

- Circumscribed labyrinthitis
- Diffuse serous labyrinthitis
- Diffuse suppurative labyrinthitis

Circumscribed labyrinthitis (Fistula of labyrinth)	Diffuse serous labyrinthitis	Diffuse suppurative labyrinthitis
<ul style="list-style-type: none"> ♦ Localized perilyabyrinthitis following inflammatory condition where pathological fistula formation occurs mainly into lateral semicircular canal ♦ Also called paralabyrinthitis 	<ul style="list-style-type: none"> ♦ Diffuse labyrinthine infection without pus formation 	<ul style="list-style-type: none"> ♦ Diffuse pyogenic infection of labyrinth with permanent loss of vestibular and cochlear function

Etiology

<ul style="list-style-type: none"> ♦ Cholesteatoma (MC) ♦ Chronic otitis media (acute exacerbation) ♦ Acute coalescent mastoiditis ♦ Middle ear or temporal bone tumors like carcinoma or glomus tumor ♦ Surgical or accidental trauma to labyrinth ♦ Congenital syphilis ♦ Granulomatous diseases 	<ul style="list-style-type: none"> ♦ Preexisting circumscribed labyrinthitis ♦ CSOM or cholesteatoma—untreated ♦ Acute middle ear infection ♦ Mastoidectomy in presence of circumscribed labyrinthitis ♦ Trauma ♦ Surgery of inner ear like stapedectomy or fenestration operation 	<ul style="list-style-type: none"> ♦ Preexisting diffuse serous labyrinthitis ♦ Chronic suppurative otitis media ♦ Subdural abscess or meningitis
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Pathogenesis

Primary breakdown of middle ear mucosa ↓ Resorption of bone along vascular channels ↓ Resorption of portion of labyrinthine capsule ↓ Fistula formation ↓ Labyrinthitis	Toxic or suppurative process on membrane barrier of labyrinth ↓ Bony fistula ↓ Chemical changes in perilymphatic space ↓ Cellular infiltration of serous or serofibrinous exudates	a. Acute stage <ul style="list-style-type: none"> – Consists of infiltration of labyrinth with polymorphonuclear leukocytes combined with destruction of soft tissues – Osseous labyrinth becomes necrosed in parts with granulation tissue forming wall around necrotic bone b. Chronic stage <ul style="list-style-type: none"> – 2–3 weeks after acute stage – Granulation tissue gradually changes to fibrous tissue associated with calcification and new bone formation
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<i>Circumscribed labyrinthitis (Fistula of labyrinth)</i>	<i>Diffuse serous labyrinthitis</i>	<i>Diffuse suppurative labyrinthitis</i>
<i>Clinical features</i>		
<i>Symptoms</i> <ul style="list-style-type: none"> ♦ Vertigo—transient and often induced by pressure on tragus, cleaning ear or performing Valsalva maneuver ♦ Nausea <i>Signs</i> <ul style="list-style-type: none"> ♦ Spontaneous nystagmus with quick component towards affect ear ♦ Fistula test positive (feeling of vertigo on application of positive pressure in ear canal) 	<i>Symptoms</i> <ul style="list-style-type: none"> ♦ Vertigo (mild to severe)—spontaneous and rotational ♦ Nausea and vomiting ♦ Difficulty in hearing <i>Signs</i> <ul style="list-style-type: none"> ♦ Spontaneous nystagmus with quick component towards affected ear ♦ Fistula test positive if secondary to circumscribed labyrinthitis ♦ Sensorineural hearing loss - acute onset but temporary 	<ul style="list-style-type: none"> ♦ Onset may be acute (in acute) or insidious (in chronic) <i>Symptoms</i> <ul style="list-style-type: none"> ♦ Vertigo—severe but relieved after 3–6 weeks due to adaptation by central mechanism ♦ Nausea and vomiting ♦ Ataxia ♦ Dizziness ♦ Loss of balance ♦ Total loss of hearing <i>Signs</i> <ul style="list-style-type: none"> ♦ Toxic appearance ♦ Spontaneous nystagmus (horizontal or rotatory type) with quick component towards healthy side ♦ Fistula test negative (due to dead labyrinth) ♦ Profound sensorineural deafness
<i>Investigations</i>		
		<ul style="list-style-type: none"> ♦ Caloric test—canal paresis ♦ Audiogram—profound sensorineural hearing loss ♦ Radiography—CT scan
<i>Treatment</i>		
<i>Conservative</i> <ol style="list-style-type: none"> Antibiotics <ul style="list-style-type: none"> – To control and prevent spread of infection Labyrinthine sedatives <ul style="list-style-type: none"> – Prochlorperazine or dimenhydrinate for symptomatic relief of vertigo <i>Operative</i> <ol style="list-style-type: none"> Mastoidectomy and closure of fistula <ul style="list-style-type: none"> – In CSOM and cholesteatoma 	<i>Conservative</i> <ol style="list-style-type: none"> Absolute bed rest <ul style="list-style-type: none"> – Immobilization of patient in bed with affect ear above Antibiotics <ul style="list-style-type: none"> – To control infection Labyrinthine sedatives <ul style="list-style-type: none"> – Prochlorperazine or dimenhydrinate for symptomatic relief of vertigo <i>Operative</i> <ul style="list-style-type: none"> ♦ C/I in early stage <i>Indication</i> <ul style="list-style-type: none"> ♦ Nonresponsive to conservative treatment ♦ Cholesteatoma <ol style="list-style-type: none"> Mastoidectomy <ul style="list-style-type: none"> – Cortical (acute mastoiditis) or Modified radical mastoidectomy (CSOM with cholesteatoma) 	<i>Conservative</i> <ol style="list-style-type: none"> Absolute bed rest <ul style="list-style-type: none"> – Immobilization of patient in bed with affect ear above Antibiotics <ul style="list-style-type: none"> – To control infection Labyrinthine sedatives <ul style="list-style-type: none"> – Prochlorperazine or dimenhydrinate for symptomatic relief of vertigo <i>Operative</i> <ol style="list-style-type: none"> Mastoidectomy <ul style="list-style-type: none"> – Cortical (acute mastoiditis) or Modified radical mastoidectomy (CSOM with cholesteatoma) Labyrinthine drainage <ul style="list-style-type: none"> – If intralabyrinthine suppuration acts as source of intracranial complication

4. Vestibular function tests.

Refer Question No. 3 June 2009 (RS2).

5. Pathophysiology of nasal allergy.

- Allergic rhinitis is an IgE-mediated immunological response of nasal mucosa to airborne allergens.

Etiology	Predisposing factors
i. Inhalant allergens (most common) <ul style="list-style-type: none"> – Pollens from trees and grasses – Mould spores – House dust – Debris from insects or house mite ii. Ingestant allergen <ul style="list-style-type: none"> – Foods like eggs, fish, milk, etc. iii. Contactant allergen <ul style="list-style-type: none"> – Cosmetics and powders iv. Irritants <ul style="list-style-type: none"> – Fumes and smoke v. Drugs <ul style="list-style-type: none"> – Aspirin, antihypertensives, iodides vi. Infections <ul style="list-style-type: none"> – Bacterial or protozoal infections 	<ul style="list-style-type: none"> ♦ Heredity (increased risk if either or both parents affected) ♦ Hormonal changes (during pregnancy, puberty) ♦ Climate change (humidity, environmental pollution) ♦ Psychological

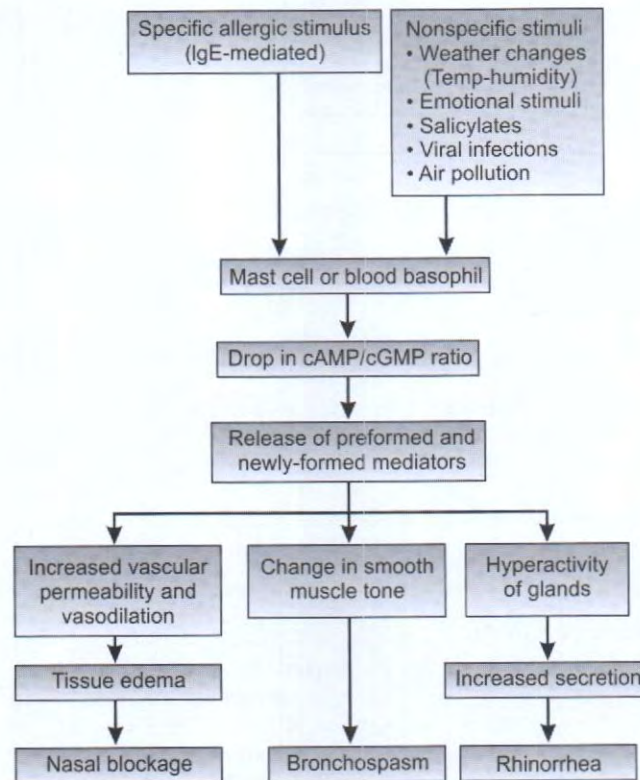


Figure 11: Allergic rhinitis—etiopathogenesis

Pathophysiology of Nasal Allergy

Primary response (priming)	Secondary response		
♦ First exposure to allergen induces production of specific IgE antibody in genetically predisposed individuals which gets fixed to blood basophils or tissue mast cells by its Fc end	♦ On subsequent exposure, allergen combines with IgE antibody at its Fab end causing degranulation of mast cells, releasing several chemical mediators		
	Acute / Early phase	Late / delayed phase	Inflammatory phase
	♦ Occurs immediately or within 5–30 minutes ♦ Mediated by vasoactive amines like histamine	♦ Occurs 2–8 hours after exposure to allergen without additional exposure ♦ Mediated by PGs, slow reacting substances of anaphylaxis (SRS-A), TXA and neutrophils	♦ Due to infiltration of inflammatory cells, i.e. eosinophils, neutrophils, basophils, monocytes and CD4+ T cells at site of antigen deposition

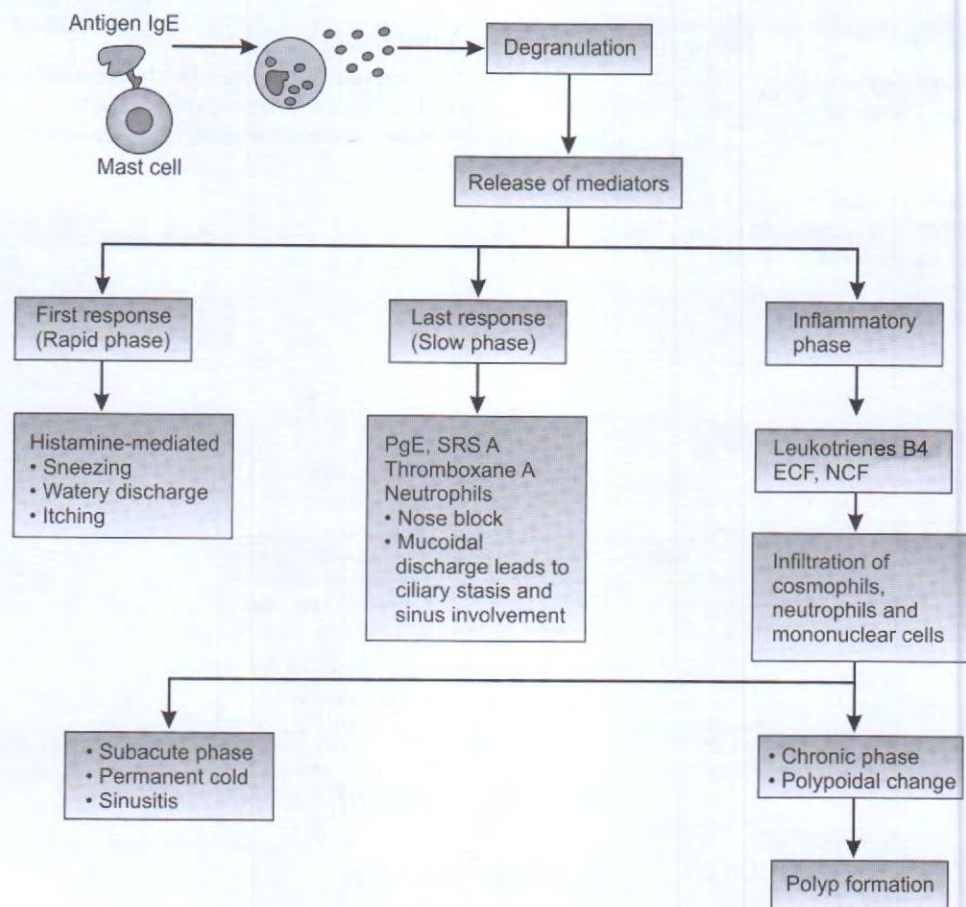


Figure 12: Allergic rhinitis—pathogenesis

Clinical Features

- Usual age of onset 12–16 years and recedes after 40–50 years of age
- May be seasonal or perennial.

Symptoms		Signs
Seasonal allergic rhinitis	Perennial allergic rhinitis	
<ul style="list-style-type: none"> ♦ Paroxysmal sneezing of recurrent type (exhausts patient) ♦ Nasal obstruction ♦ Thin watery and copious nasal discharge ♦ Itching in nose, eyes, palate or pharynx ♦ Bronchospasm (in some) 	<ul style="list-style-type: none"> ♦ Frequent colds ♦ Persistently stuffy nose ♦ Thin watery or mucoid nasal discharge ♦ Loss of smell sensation ♦ Postnasal drip ♦ Mouth breathing ♦ Chronic cough ♦ Hearing impairment (due to blockage of Eustachian tube or fluid in middle ear) 	<p>a. Nasal</p> <ul style="list-style-type: none"> – Darrier's line (a transverse black nasal crease across middle of dorsum of nose a little superior to tip of nose due to constant upward rubbing of nose simulating a salute—allergic salute) – Pale, edematous nasal mucosa in acute stage but later bluish in chronic stage – Hypertrophied turbinates <p>b. Ocular</p> <ul style="list-style-type: none"> – Edema of lids – Congestion and cobble stone appearance of conjunctiva – Dark circles under eye (allergic shiners due to venous stasis) <p>c. Otological (due to blockage of Eustachian tube)</p> <ul style="list-style-type: none"> – Retracted tympanic membrane – Serous otitis media <p>d. Pharyngeal</p> <ul style="list-style-type: none"> – Granular pharyngitis (hyperplasia of submucosal lymphoid tissue) <p>e. Laryngeal</p> <ul style="list-style-type: none"> – Hoarseness of voice – Vocal cord edema

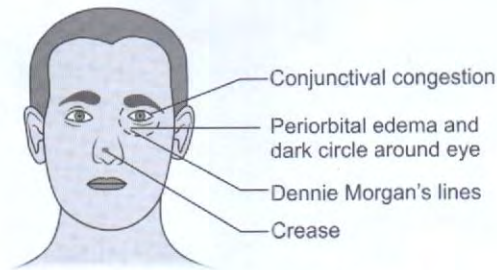


Figure 13: Signs of allergic rhinitis

Investigations

TLC and DLC	Nasal smear	Skin tests	Radioallergosorbent test (RAST)	Nasal provocation test
♦ Reveals peripheral eosinophilia	♦ Demonstrates large number of eosinophils (taken during clinically active disease)	♦ Identifies specific allergen (prick, scratch and intradermal)	♦ In-vitro test measuring concentration of specific IgE antibody	♦ Crude method of testing allergy to specific allergen by asking to sniff allergen and observing for allergic symptoms

Treatment

Conservative

- a. Avoidance of allergen
 - Ideal but not practical
 - Most successful if involves single antigen
 - Example: Removal of pet, encasing pillow or mattress with plastic sheet, change of place of work or change of job.
- b. Pharmacotherapy
 - i. Antihistamines
 - Control rhinorrhea, sneezing and pruritus
 - Example: Cetrizine, fexofenadine, loratadine.
 - ii. Sympathomimetic drugs (oral or topical)
 - α -adrenergic drugs to constrict blood vessels and reduce nasal congestion and edema
 - Example: Phenylephrine, oxymetazoline and xylometazoline.
 - iii. Corticosteroids
 - Topical sprays
 - * Inhibit recruitment of inflammatory cells into nasal mucosa and suppress late phase allergic reaction
 - * Example: Beclomethasone dipropionate, budesonide.
 - Oral
 - * To control symptoms but only during acute episodes
 - * Example: Hydrocortisone, dexamethasone.
 - Topical injection
 - * 0.5 mL of hydrocortisone injected in inferior turbinate on either side once a week for a month offers relief for about a year.
 - iv. Mast cell stabilizers
 - To stabilize mast cells and prevents them from degranulation despite formation of IgE - antigen complex
 - Example: Sodium chromoglycate as 2% solution for nasal drops/spray/aerosol powder.
- c. Chemical cautery
 - Application of 15% silver nitrate to anterior part of inferior turbinate once a week for a month after local anesthesia.
- d. Immunotherapy
 - Indicated in cases of failure of pharmacotherapy and adverse effects of drugs
 - Consists of administering gradually increasing dose of allergen till maintenance dose is reached
 - Suppresses formation of IgE and raises titre of specific IgG antibody.

Operative

- Removal of nasal obstruction by removal of polyp, correction of DNS, or hypertrophied turbinates
- Removal of septic foci.

Complications

- Recurrent sinusitis (due to obstruction of sinus ostia)
- Nasal polyposis
- Serous otitis media
- Orthodontic problems in children (due to prolonged mouth breathing)
- Bronchial asthma.

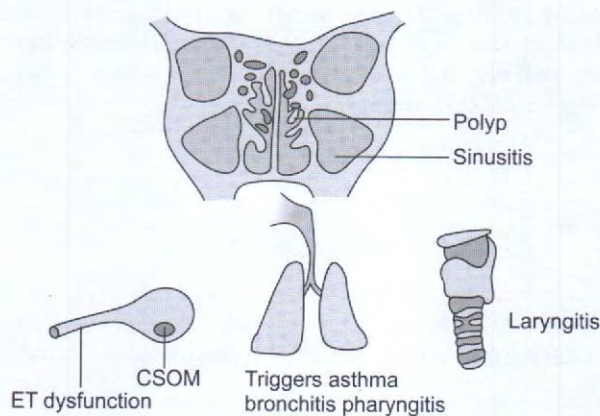


Figure 14: Complications of allergic rhinitis

6. Le Fort fractures.

Refer Question No. 3 December 2010 (RS2).

7. Congenital laryngeal stridor.

- Laryngomalacia is most common congenital anomaly of larynx
- Also called congenital laryngeal stridor because it is most common cause of congenital stridor.

Etiopathogenesis

- Excessive flaccidity of structures bounding supraglottic larynx particularly aryepiglottic fold → sucking in of these during inspiration → laryngeal narrowing → inspiratory stridor.

Pathology

- Excessive softness, flabbiness or lack of consistency of laryngeal tissue
- Thinning and hypocellularity of laryngeal cartilages
- Wrinkled loose edematous mucosa.

Clinical Features

- Manifests at birth or soon after.

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Stridor, increased on crying or exertion but subsides on placing child in prone position ♦ Cyanosis (sometimes) 	<ul style="list-style-type: none"> ♦ Elongated, tubular epiglottis, curled upon itself (omega-shaped) ♦ Floppy aryepiglottic folds ♦ Prominent arytenoids ♦ Cruciate appearance of laryngeal inlet (due to prolapsed of laryngeal soft tissue into vestibule)



Figure 15: Laryngomalacia

Treatment

Conservative	Operative
<ul style="list-style-type: none"> ♦ Reassurance ♦ Avoid rough handling of children, sudden shocks to them or changes in temperature 	<ul style="list-style-type: none"> ♦ Tracheostomy in severe respiratory obstruction

Prognosis

- Spontaneous regression by 2 years of age.

8. Fungal sinusitis.

- Fungal sinusitis is the inflammation of sinus mucosa due to fungal infection.

Etiology

Causative organisms	Predisposing factors
<ul style="list-style-type: none"> ♦ Aspergillus (most common) ♦ Mucormycosis ♦ Pecilomyces ♦ Candida ♦ Penicillium ♦ Rhizopus 	<ul style="list-style-type: none"> ♦ Immunocompromized patients (diabetes, HIV, etc.) ♦ Prolonged steroidal nasal spray use ♦ Occupations like farmers, etc.

Pathology (Types)

Mycetoma fungal sinusitis	Allergic fungal sinusitis	Chronic indolent sinusitis	Fulminant sinusitis
Fungal ball (clumps of spores) in sinus cavity	Allergic reaction to fungal spores dispersed in air	Invasive form characterized by nodular-shaped granulomatous inflammatory lesions	Characterized by progressive destruction of sinuses and intra or extracranial complications

Clinical Features

- Mostly affects maxillary sinus
- Progresses from months to years (depending upon age and immune status of patient)

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Thick purulent nasal discharge, occasionally blood-stained ♦ Nasal obstruction ♦ Epistaxis ♦ Bad smell from nose (halitosis) ♦ Chronic headache ♦ Diplopia and visual impairment 	<ul style="list-style-type: none"> ♦ Swelling of cheek or orbit ♦ Proptosis ♦ Unilateral polyposis ♦ Mass in middle meatus with purulent discharge

Investigations

a. Diagnostic nasal endoscopy	♦ To locate lesion and take biopsy
b. Nasal swab	♦ For fungal culture
c. Biopsy	♦ Histopathological examination for identifying causative agent
d. Radiology	<ul style="list-style-type: none"> ♦ X-rays, CT scan, MRI shows hyperdense areas, hypertrophied mucosa and areas of hyperattenuation due to calcareous deposits ♦ Bony erosions may be seen in indolent and intracranial spread may be seen in fulminant form

Treatment

Conservative	Operative
a. Antifungal drugs for up to 12 months <ul style="list-style-type: none"> – Itraconazole—for aspergillus, palciliomyces – Fluconazole—for candida – Amphotericin B—for aspergillus, mucormycosis b. Steroids <ul style="list-style-type: none"> – Given preoperatively and postoperatively especially in fulminant type 	a. Functional endoscopic sinus surgery <ul style="list-style-type: none"> – Consists of debridement of sinus cavity of fungal material and establishment of drainage

Complications (Common in Indolent and Fulminant Form)

- Orbital cellulitis
- Orbital abscess
- Meningitis
- Brain abscess
- Cavernous sinus thrombosis.

9. Osteomeatal complex.

- Middle meatus situated on lateral wall of nose under middle turbinate is called osteomeatal complex.

Features

Bulla ethmoidalis	Uncinate process	Hiatus semilunaris	Infundibulum
<ul style="list-style-type: none"> ♦ An ethmoidal cell situated behind uncinate process ♦ May be a pneumatized cell or a solid bony prominence ♦ May extend superiorly to base of skull and posteriorly fuse with ground lamella ♦ Space above or behind bulla is respectively called suprabulbar or retrobulbar recesses which together form lateral sinus of Grunwald 	<ul style="list-style-type: none"> ♦ A hook like structure running in from anterosuperior to posteroinferior direction ♦ Presents sharp posterosuperior border running parallel to anterior border of bulla ethmoidalis, enclosing hiatus semilunaris in between them ♦ Anteroinferior border is attached to lateral wall ♦ Posteroinferior end is attached to inferior turbinate dividing membranous part lower middle meatus into anterior and posterior fontanelle 	<ul style="list-style-type: none"> ♦ 2 dimensional semicircular sulcus running between anterior border of bulla ethmoidalis and posterosuperior border of uncinate process ♦ Measures only 1–2 in width 	<ul style="list-style-type: none"> ♦ Small funnel-shaped passage anterior to bulla ♦ Limited medially by uncinate process and frontal process of maxilla and sometimes by lacrimal bone ♦ Bound laterally by lamina papyracea

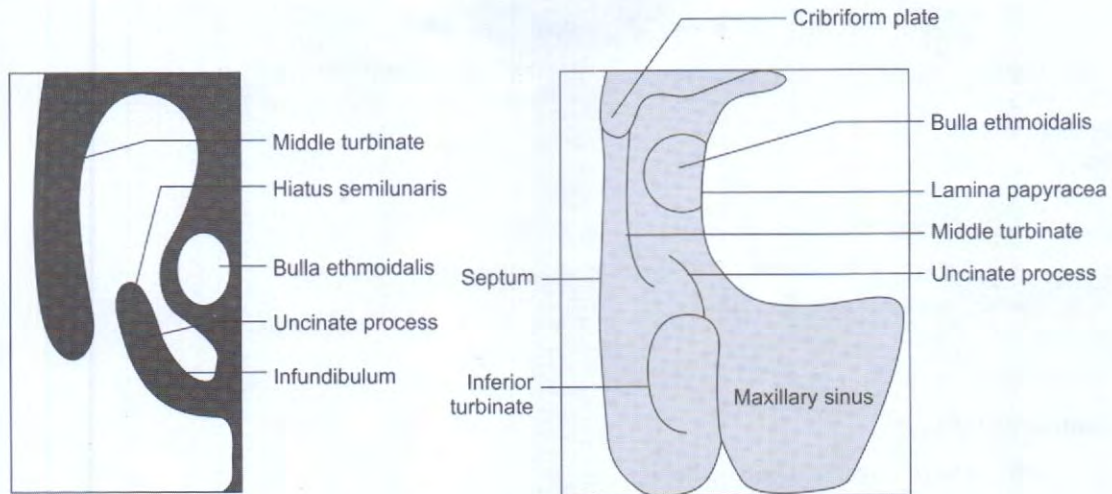


Figure 16: Osteomeatal complex

Openings

Frontal sinus (variable depending upon attachment of upper end of uninate process)	Anterior and middle ethmoidal sinuses	Maxillary sinus
<ul style="list-style-type: none"> ♦ Frontal recess (55%) ♦ Above infundibulum (30%) ♦ Into infundibulum (15%) ♦ Above bulla ethmoidalis (1%) 	<ul style="list-style-type: none"> ♦ Middle meatus 	<ul style="list-style-type: none"> ♦ Posterior part of infundibulum (hiatus semilunaris) ♦ Accessory opening of maxillary sinus (30%) ♦ Anterior or posterior fontanel

Applied Anatomy

- Middle meatus shows several important structures and openings of numerous paranasal sinuses which are important in endoscopic sinus surgery.

10. Fibrous Dysplasia.

- Fibrous dysplasia is benign neoplasm of paranasal air sinuses
- Mostly involves maxillary air sinus but sometimes also involves ethmoidal and frontal sinuses.

Etiopathogenesis

Etiology	Pathology
<ul style="list-style-type: none"> ♦ Idiopathic 	<ul style="list-style-type: none"> ♦ Replacement of normal medullary bone by collagen, fibroblasts and varying amounts of osteoid tissue

Types

Monostatic	Polyostotic	Craniofacial	Cherubism
Involves one bone	Involves multiple bones	Involvement of cranium and facial bones	Familial variant

Clinical Features

- Usually seen at puberty and ceases by 20–25 years
- More common in females.

Symptoms

- ♦ Disfigurement of face
- ♦ Nasal obstruction

Signs

- ♦ Bony hard, diffuse, painless swelling
- ♦ Proptosis

Investigations

- | | |
|------------|--|
| a. X-rays | ♦ Ground glass appearance of bone |
| b. CT scan | ♦ Obliteration of sinuses with obliteration of infraorbital margin |
| c. Biopsy | ♦ Reveals fibrous and osseous tissue |

Treatment

Operative (treatment of choice)

- Surgical resculpting or removal of involved bone.

11. Management of Otospongiosis.

Refer Question No. 1 December 2010 (RS2).

12. Noise-induced hearing loss.

- Noise-induced hearing loss is an occupation disorder seen commonly in workers employed in noisy factories.

Etiology

Etiology	Predisposing factors
<ul style="list-style-type: none"> ♦ Acute exposure to intense sound (acoustic trauma) ♦ Chronic exposure to less intense sound 	<ul style="list-style-type: none"> ♦ Exposure to frequency in range of 2000–3000 Hz ♦ Exposure to high intensity sound beyond maximum permissible limit (legal limit —90 dB 8 hours/day for 5 days/week) ♦ Continuous exposure rather than intermittent ♦ Preexisting ear disease

Types

Temporary threshold shift	Permanent threshold shift
<ul style="list-style-type: none"> ♦ Impaired hearing immediately after exposure to noise but recovers after an interval of few minutes to few hours ♦ Due to depletion of metabolic enzymes and diminished oxygen tension 	<ul style="list-style-type: none"> ♦ Permanent impairment of hearing with no recovery ♦ Due to irreversible morphological changes in hair cells

Pathology

- Damage to hair cells, starting in base turn of cochlea, affecting outer hair cells before inner hair cells.

Clinical Features

- Bilaterally symmetrical.

Symptoms

- Difficulty in hearing in noisy environment
- High-pitched tinnitus
- Difficulty in understanding speech.

Investigations

- Audiogram
 - Acoustic dip, i.e. characteristic notch at 4000 Hz for both air and bone conduction initially but later deepens and widens to involve lower and higher frequencies.

(Notch is seen characteristically at 4000 Hz because it corresponds anatomically to area of basal turn of cochlea where basilar membrane is firmly attached to it thus more prone for torsion and pressure changes in perilymph)

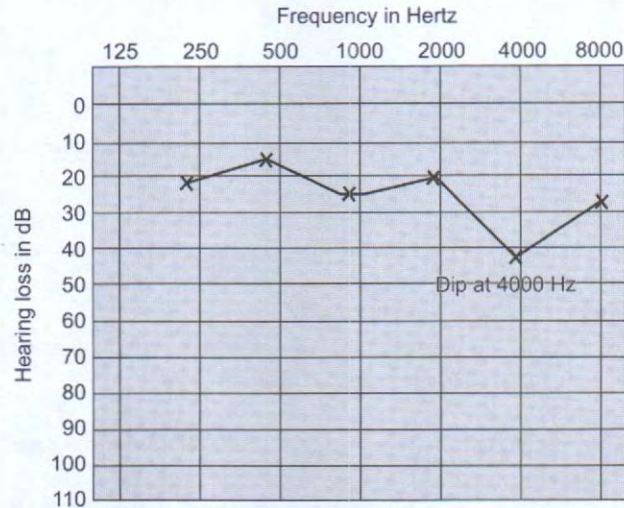


Figure 17: Noise-induced hearing loss—audiogram

Prophylaxis

- Preplacement and periodic examination
- Ear plugs or muffs (provide protection up to 35 dB).

Treatment

- Hearing aids or cochlear implants.

SHORT ANSWERS

13. Tympanic plexus.

- Tympanic plexus is a neural plexus located in middle ear cavity.

Location	Formation	Area supplied
<ul style="list-style-type: none"> ♦ On promontory on medial wall of middle ear 	<ul style="list-style-type: none"> ♦ Tympanic branch of glossopharyngeal nerve ♦ Sympathetic fibers from plexus around internal carotid artery 	<ul style="list-style-type: none"> ♦ Medial surface of tympanic membrane ♦ Middle ear cavity ♦ Mastoid air cells ♦ Eustachian tube (Bony part)

Applied Anatomy

- Tympanic plexus also supplied secretomotor fibers to parotid gland
- In Frey's syndrome, tympanic branch of glossopharyngeal nerve can be sectioned in middle ear.

14. Blood supply of tonsil.

- Palatine tonsils are a pair of almond-shaped masses of lymphoid tissue situated bilaterally in lateral wall of oropharynx.

Situation

- Triangular tonsillar sinus bounded in front by palatoglossal fold, behind by palatopharyngeal fold and base by dorsal surface of posterior 1/3rd of tongue.

Morphology

- Two surfaces (medial and lateral)
- Two borders (anterior and posterior)
- Two poles (upper and lower)

Medial surface	<ul style="list-style-type: none"> ♦ Free and bulges into oropharynx ♦ Lined by nonkeratinized stratified squamous epithelium continuous with that of mouth <p><i>Features</i></p> <ol style="list-style-type: none"> Tonsillar pits <ul style="list-style-type: none"> – Small openings about 12–15, leading into a mucus tubule known as tonsillar crypt surrounded by numerous lymphatic follicles Intratonsillar cleft (largest crypt of the tonsil) <ul style="list-style-type: none"> – Deep semilunar fissure affecting upper part of tonsil representing remnant of second branchial pouch Embryonic folds (replaced by the lymphoid tissue after birth) <ul style="list-style-type: none"> – Plica triangularis <ul style="list-style-type: none"> – Triangular estigial fold of mucus extending backwards from lower part of palatoglossal arch – Plica semilunaris <ul style="list-style-type: none"> – Semilunar fold of mucosa crossing upper part of tonsil, arching backward from upper part of palatoglossal arch
Lateral or deep surface	<ul style="list-style-type: none"> ♦ Extends above, below and in front beyond limits of tonsillar sinus ♦ Covered by a sheet of fascia which is an extension of pharyngobasilar fascia and forms capsule of tonsil ♦ Capsule is only loosely attached to muscular wall of pharynx but anteroinferiorly is firmly adherent to sides of tongue just in front of insertion of palatoglossus and palatopharyngeous muscles ♦ This firm attachment helps to keep tonsil in place during swallowing <p><i>Relations (Tonsil bed) from within outward</i></p> <ol style="list-style-type: none"> Loose areolar tissue containing paratonsillar veins Pharyngobasilar fascia Superior constrictor muscle of pharynx Buccopharyngeal fascia, containing pharyngeal plexus of nerves and vessels Arteries <ul style="list-style-type: none"> – Facial artery with its ascending palatine and tonsilar branches – Ascending pharyngeal artery – Internal carotid artery lying about 2.5 cm behind and lateral to tonsillar sinus and is separated by fibrofatty tissue Styloglossus, stylopharyngeus and glossopharyngeal nerve Posterior belly of digastric and stylohyoid muscles Medial pterygoid muscle and ramus of mandible
Anterior border	♦ Related to palatoglossal arch with its muscle
Posterior border	♦ Related to palatopharyngeal arch with its muscle
Upper pole	♦ Related to soft palate
Lower pole	♦ Continues with lingual tonsil and is connected to side of tongue by a band of fibrous tissue known as suspensory ligament of tonsil which helps to keep tonsil in position

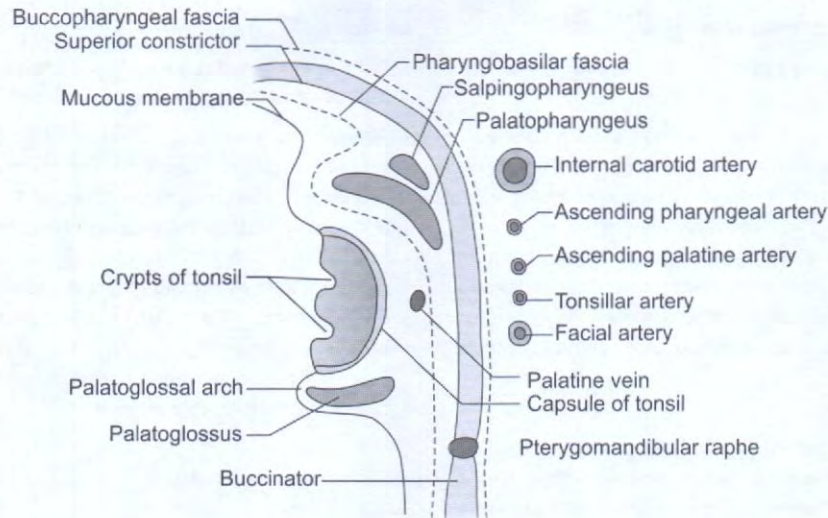


Figure 18: Palatine tonsil—medial and lateral relations

Arterial supply	Venous drainage	Lymphatic drainage	Nerve supply
<p>a. Main</p> <ul style="list-style-type: none"> – Tonsillar branch of facial artery <p>b. Additional</p> <ul style="list-style-type: none"> – Ascending palatine branch of facial artery – Dorsal lingual branches of lingual artery – Ascending pharyngeal branch of external carotid artery – Greater palatine branch of maxillary artery 	<ul style="list-style-type: none"> ♦ Through one or more veins leaving lower part of lateral surface into palatine, pharyngeal or facial veins 	<ul style="list-style-type: none"> ♦ Into jugulo – digastric nodes 	<ul style="list-style-type: none"> ♦ Glossopharyngeal and lesser palatine nerves

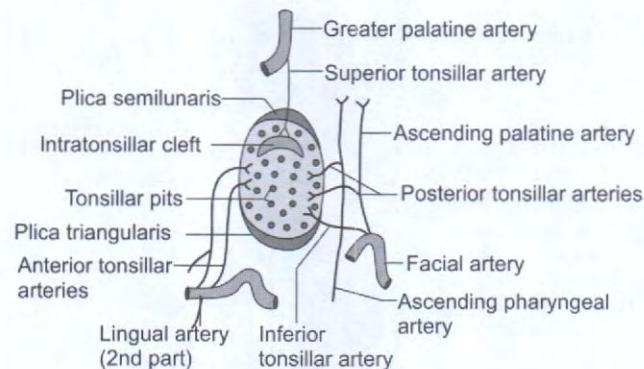


Figure 19: Palatine tonsil—arterial supply

Development

- Palatine tonsil develops in relation to lateral parts of 2nd pharyngeal pouch
- Endoderm lining 2nd pharyngeal pouch undergoes considerable proliferation thus obliterating most of the pouch
- There is collection of lymphocytes which develop in situ or are derived from blood in relation to these endodermal cells
- Persisting part of 2nd pharyngeal pouch is represented as intratonsillar cleft or tonsillar fossa.

15. Recurrent laryngeal nerve.

- Recurrent laryngeal nerves are pair of branches of vagus nerve
- They are so called because they take a circuitous route up to thorax and ascend back up in neck to supply larynx.

	<i>Right recurrent laryngeal nerve</i>	<i>Left recurrent laryngeal nerve</i>
Origin	♦ From right vagus in front of right subclavian artery	♦ From left vagus in thorax when it cross anterior and left surface of arch of aorta
Course	♦ After origin, nerve winds backwards below subclavian artery and runs upward and medially behind subclavian and common carotid arteries to reach tracheoesophageal groove ♦ In upper part of groove, it is related to inferior thyroid artery either lying superficial or deep to it. Nerve then passes deep to lower border of inferior constrictor and enters larynx behind cricothyroid joint	♦ After origin, nerve winds round undersurface of arch of aorta, behind and to left side of ligamentum arteriosum and passes upward at first between bifurcation of trachea and aortic arches. As it ascends, it lodges in left tracheoesophageal groove and appears in neck ♦ In neck, it runs medially to left common carotid artery. At lower end of lateral lobe of thyroid gland, it usually lies posterior to loop of inferior thyroid artery. Further upward, it occupies medial surface of thyroid gland either medial or lateral to ligament of Berry. Nerve then passes deep to lower border of inferior constrictor and enters larynx behind cricothyroid joint
Branches	♦ Cardiac branches form deep cardiac plexus ♦ Communicating branch joins with inferior cervical ganglion of sympathetic trunk ♦ Branches to trachea and esophagus supply muscles, glands and mucosa ♦ A motor twig to inferior constrictor muscle ♦ Articular branches to cricothyroid and cricoarythenoid joints ♦ Muscular branches to all intrinsic muscles of larynx except cricothyroid ♦ Sensory branches to mucus membrane of larynx below the vocal folds	
Applied anatomy	♦ Recurrent laryngeal nerves may be injured in thyroid surgery or compressed by a growing tumor, aortic aneurysm or from any other causes ♦ Right recurrent laryngeal nerve is more susceptible to damage during thyroid surgery due to its relatively medial location ♦ If damage is unilateral, patient may present with voice changes including hoarseness ♦ Bilateral nerve damage can result in breathing difficulties and aphonia	

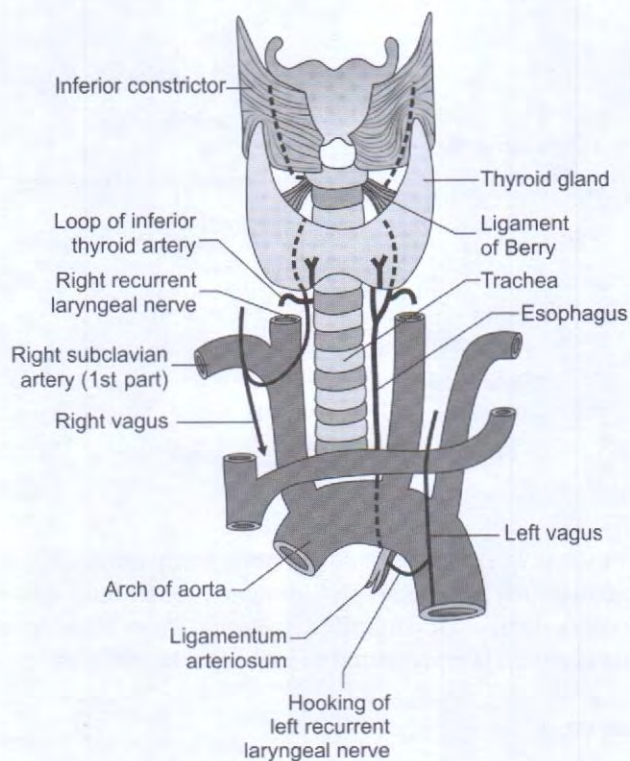


Figure 20: Recurrent laryngeal nerve—course

16. Eustachian tube.

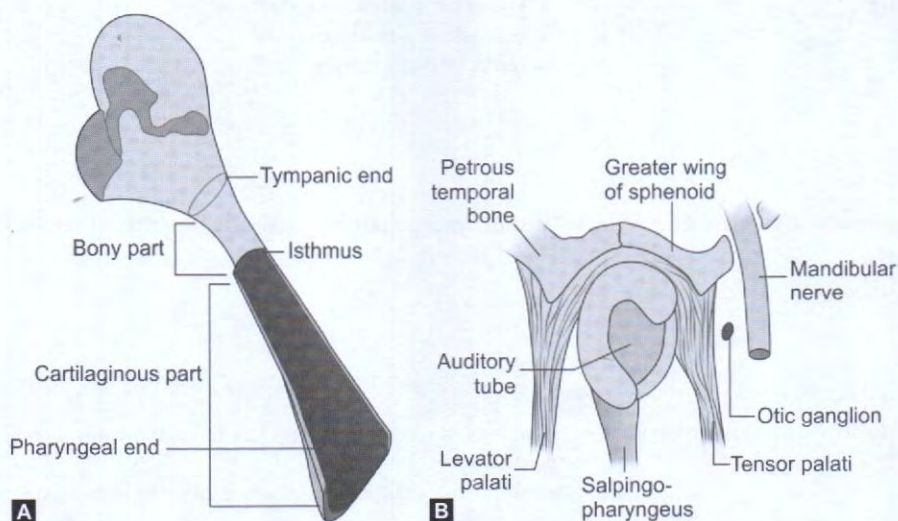
- Eustachian tube is mucus-lined ossocartilaginous channel which communicates lateral wall of nasopharynx with anterior wall of middle ear
- Also called auditory tube or pharyngotympanic tube
- It usually remains closed and opens during swallowing, yawning and sneezing by action of tensor and levator palati.

Dimensions

- 36 mm long
- Directed downward, forward and medially forming an angle of 45° with sagittal plane and 30° with horizontal plane.

Parts

Bony part	Cartilagenous part
<ul style="list-style-type: none"> ♦ 12 mm long part forming posterior and lateral 1/3rd of tube ♦ Lies in petrous temporal bone near tympanic plate ♦ Wide lateral end opens on anterior wall of middle ear cavity and narrow medial end (isthmus) is jagged for attachment of cartilagenous part 	<ul style="list-style-type: none"> ♦ 24 cm long part forming anterior and medial 2/3rd of tube ♦ Lies in sulcus tubae ♦ Its superior and medial wall is formed by a triangular plate of cartilage and lateral wall and floor is formed by a fibrous membrane ♦ Apex is attached to bony part and free base forms a tubal elevation called torus tubarius in nasopharynx 1–1.25 cm behind posterior end of inferior turbinate
Relations	
<p><i>Superior</i></p> <ul style="list-style-type: none"> ♦ Canal for tensor tympani <p><i>Medial</i></p> <ul style="list-style-type: none"> ♦ Carotid canal <p><i>Lateral</i></p> <ul style="list-style-type: none"> ♦ Chorda tympani, ♦ Spine of sphenoid ♦ TM joint <p><i>Inferior</i></p> <ul style="list-style-type: none"> ♦ Tympanic plate of temporal bone 	<p><i>Anterolaterally</i></p> <ul style="list-style-type: none"> ♦ Tensor palati, Mandibular nerve and its branches, Otic ganglion, Chorda tympani, Middle meningeal artery and Medial pterygoid plate <p><i>Posteromedially</i></p> <ul style="list-style-type: none"> ♦ Petrous temporal and Levator palati <p><i>Superiorly</i></p> <ul style="list-style-type: none"> ♦ Sulcus tubae of skull base <p><i>Inferiorly</i></p> <ul style="list-style-type: none"> ♦ Superior constrictor muscle, Processus tuberosus, a few fibers of tensor veli palatine



Figures 21A and B: (A) Auditory tube—parts; (B) Cartilagenous part of auditory tube—relations

Arterial supply	Venous drainage	Nerve supply
<ul style="list-style-type: none"> ♦ Ascending pharyngeal artery ♦ Middle meningeal arteries ♦ Artery of Pterygoid canal 	<ul style="list-style-type: none"> ♦ Into pharyngeal and pterygoid plexus of veins 	<ul style="list-style-type: none"> ♦ At ostium: Pharyngeal branch of pterygopalatine ganglion (maxillary nerve) ♦ Cartilaginous part: Nervus spinosus (mandibular nerve) ♦ Bony part: Tympanic plexus (glossopharyngeal nerve)

Functions

- Ventilation and equalization of air pressure in middle ear cavity with atmospheric pressure
- Protection against nasopharyngeal sound pressure and reflux nasopharyngeal secretions
- Clearance of middle ear secretions.

Applied Anatomy

- Auditory tube can act as a portal of entry for infection from nasopharynx to middle ear cavity which is more common in children because tube is shorter and straighter in them and opens at lower level
- Inflammation of tubal tonsil may block tube causing retraction of tympanic membrane producing disturbance of hearing.

17. Traumatic perforation of tympanic membrane.

- Traumatic perforation of tympanic membrane is very common but self-healing if treated early.

Etiology

- Trauma due to pointed foreign bodies like hair pin, matchstick, or unskilled attempt to removal foreign body
- Sudden change in air pressure as in slap or kiss on ear, sudden blast, loss pressure in aircraft, forceful blowing of nose
- Pressure by fluid column as in diving, water sports, forceful syringing
- Fracture of temporal bone.

Clinical Features

Symptoms	Signs
<ul style="list-style-type: none"> ♦ History of trauma to ear ♦ Pain ♦ Deafness ♦ Blood-stained discharge ♦ Tinnitus ♦ Vertigo 	<ul style="list-style-type: none"> ♦ Fresh bleeding or clot in external auditory canal ♦ Irregular perforation in centre of tympanic membrane with ragged inverted edges ♦ Ecchymosis of tympanic membrane ♦ Conductive hearing loss ♦ Ossicular discontinuity

Treatment

Conservative

- Packing of external ear with sterile cotton plug (if seen immediately)—usually heals within 6–8 weeks
- Avoid topical drops
- Antibiotics and analgesics.

Operative

	Indications	Procedure
a. Splintage	Fresh traumatic perforations	<ul style="list-style-type: none"> ♦ Eversion of ragged edges of perforation in tympanic membranes under operating microscope ♦ Repositioning and splinting of edges of perforation using cigarette paper, gelfilm or silicon sheet

Contd...

Contd...

	Indications	Procedure
b. Myringoplasty	Tympanic membrane perforation	<ul style="list-style-type: none"> • Incise along edges of perforation (inlay) or circumferentially inside meatus (overlay) • Place proper sized graft such that its edges extend under margins of perforation all round and a small part also covers posterior canal wall (inlay) or place graft on outer surface of tympanic membrane and make a slit in it such that it tucks under handle of malleus (overlay)
c. Cautery patching	Small, long standing central perforations with epithelialised margins	<ul style="list-style-type: none"> • Cauterize epithelialized edges of perforated tympanic membrane with 50% trichloroacetic acid • Support margins of tympanic membrane with cigarette paper moistened with 1% phenol in glycerine or steristrip, gelfoam or silicon sheets • Repeat procedure at 2 weeks intervals
d. Fat graft myringoplasty	Chronic small perforations	<ul style="list-style-type: none"> • Freshen edges of perforations using 1mm stapes hook • Scrap inside of perforation too • Insert small piece of fat harvested from ear lobule into perforation like an hour glass

Complications

- Facial paralysis
- Subluxation of stapes.

Myringoplasty

- Myringoplasty is closure of tympanic membrane perforation using graft.

Grafts used	Indications	Contraindications
<ul style="list-style-type: none"> ♦ Temporal fascia (MC) ♦ Perichondrium from tragus ♦ Tragal cartilage ♦ Vein 	<ul style="list-style-type: none"> ♦ Tympanic membrane perforation 	<ul style="list-style-type: none"> ♦ Active discharge from middle ear ♦ Nasal allergy ♦ Otitis externa ♦ Ingrowth of squamous epithelium into middle ear ♦ Deaf contralateral ear ♦ Children <3 years

Anesthesia

- Local.

Position

- Supine with face turned such that ear to be operated is superior.

Procedure

Underlay technique	Overlay technique
<ul style="list-style-type: none"> ♦ Incise along edges of perforation and removal ring of epithelium along with strip of mucosal layer from inner side of perforation ♦ Make stapes type incision and raise tympanomeatal flap to visualize integrity and mobility of ossicular chain and to assess any ingrowth of squamous epithelium into middle ear ♦ Pack middle ear with antibiotic soaked gelfoam ♦ Place proper sized graft such that its edges extend under margins of perforation all round and a small part also covers posterior canal wall ♦ Replace tympanomeatal flap 	<ul style="list-style-type: none"> ♦ Incise circumferentially inside meatus and raise meatal skin along with all epithelium from outer surface of TM remnant ♦ Place graft on outer surface of tympanic membrane and make a slit in it such that it tucks under handle of malleus ♦ Replace meatal skin, covering periphery of graft ♦ Pack ear canal with gelfoam and antibiotic pack ♦ Close endaural or postaural incision followed by matoid dressing

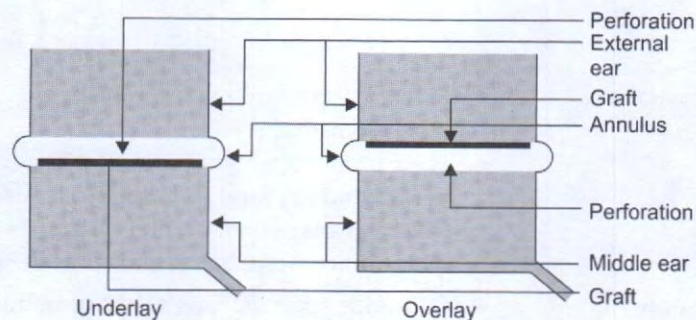


Figure 22: Myringoplasty—types

Postoperative care	Complications	
	Underlay technique	Overlay technique
<ul style="list-style-type: none"> ♦ Remove stitches after 5-6 days ♦ Remove ear pack after 5-6 days ♦ Follow-up visit at 3rd and 6th week after surgery 	<ul style="list-style-type: none"> ♦ Narrowing of middle ear ♦ Adherence of graft to promontory ♦ Anterior perforation (due to lose of graft contact with remnant of tympanic membrane) 	<ul style="list-style-type: none"> ♦ Blunting of anterior sulcus ♦ Epithelial pearls ♦ Lateralization of graft

18. Sleep apnea.

Refer Question No. 10 June 2010 (RS2).

19. Puberphonia.

- Persistence of childhood high-pitched voice in males even after puberty is called puberphonia.

Seen in	Pathophysiology
<ul style="list-style-type: none"> ♦ Emotionally immature or psychologically disturbed boys 	<ul style="list-style-type: none"> ♦ Boys with feeling of insecurity and excessive fixation to their mothers shun to assume male responsibilities

Features

- Childlike high-pitched voice
- Normal physical and sexual development.

Treatment

- Gutzmann's pressure test
 - Helps produce low-pitch voice by pressing thyroid prominence backward and downward to relax overstretched cords.

20. Gradenigo syndrome.

Refer Question No. 3 December 2009 (RS2).

21. Carcinoma in-situ.

- Presence of cytological features of malignancy but confinement of malignant cells to epithelium without invasion across basement membrane is called carcinoma in-situ or intraepithelial neoplasia (CIN)
- It is a pre-malignant lesion requiring urgent detection to prevent subsequent invasive cancer.

Examples

- Oral leukoplakia.

Etiopathogenesis

- Sequential transformation from squamous metaplasia to epithelial dysplasia to carcinoma in situ.

Features

- Characterized by morphological changes in cells, such as increased nuclear cytoplasmic ratio, pleomorphism of cells and nuclei, increased mitotic activity, poor differentiation and sometimes are accompanied by chronic inflammatory cells
- Involved area may be single and small or multifocal
- May return to normal or develop into invasive cancer.

22. Waldeyer's ring.

Refer Question No. 3 June 2015 (RS2).

MBBS PHASE III EXAMINATION

JUNE 2010
(Revised Scheme 2)

LONG ESSAYS

1. Describe the etiology, clinical features, investigations and treatment of Meniere's disease.

Refer Question No. 2 December 2008 (RS2).

2. Write about etiology, clinical features, investigations and treatment of ethmoidal polyposis.

Refer Question No. 2 December 2013 (RS2).

SHORT ESSAYS

3. Referred otalgia.

- Pain in ear (earache) is called "otalgia"
- Referred otalgia is due to causes outside of ear and referred through nerves.

Etiology

<i>Via trigeminal nerve (V nerve)—auriculotemporal branch</i>	<i>Via facial nerve (VII nerve)—greater auricular nerve</i>	<i>Via glossopharyngeal nerve (IX nerve)</i>	<i>Via vagus nerve (X nerve)</i>	<i>Via C2 and C3 spinal nerves</i>
i. Dental (Common AIM) <ul style="list-style-type: none"> – Caries – Alveolar abscess – Impacted molar – Malocclusion ii. Oral cavity (SUI) <ul style="list-style-type: none"> – Salivary calculus – Ulcerative lesions (benign/malignant) – Infection iii. TM joint <ul style="list-style-type: none"> – Arthritis – Bruxism iv. Nose, paranasal sinuses (DIM) <ul style="list-style-type: none"> – Deviated nasal septum – Infection (Sinusitis) – Malignancy v. Nasopharynx (CAN) <ul style="list-style-type: none"> – Carcinoma – Adenoidectomy – Nasopharyngitis vi. Sphenopalatine neuralgia	<ul style="list-style-type: none"> ♦ Herpes zoster ♦ Cervical spine lesions ♦ Neck lesions 	i. Tonsils <ul style="list-style-type: none"> – Acute tonsillitis – Quinsy – Post-tonsillectomy pain – Malignancy ii. Oropharynx <ul style="list-style-type: none"> – Infection (retropharyngeal and parapharyngeal abscess) – Trauma – Malignancy iii. Elongated styloid process iv. Neuralgia	i. Vallecula, epiglottis, larynx, esophagus, laryngopharynx <ul style="list-style-type: none"> – Trauma – Ulcerative lesions – Tuberculosis – Malignancy 	<ul style="list-style-type: none"> ♦ Cervical spondylolysis ♦ Cervical spine ♦ Fibrositis ♦ Myositis ♦ Trauma to cervical spine

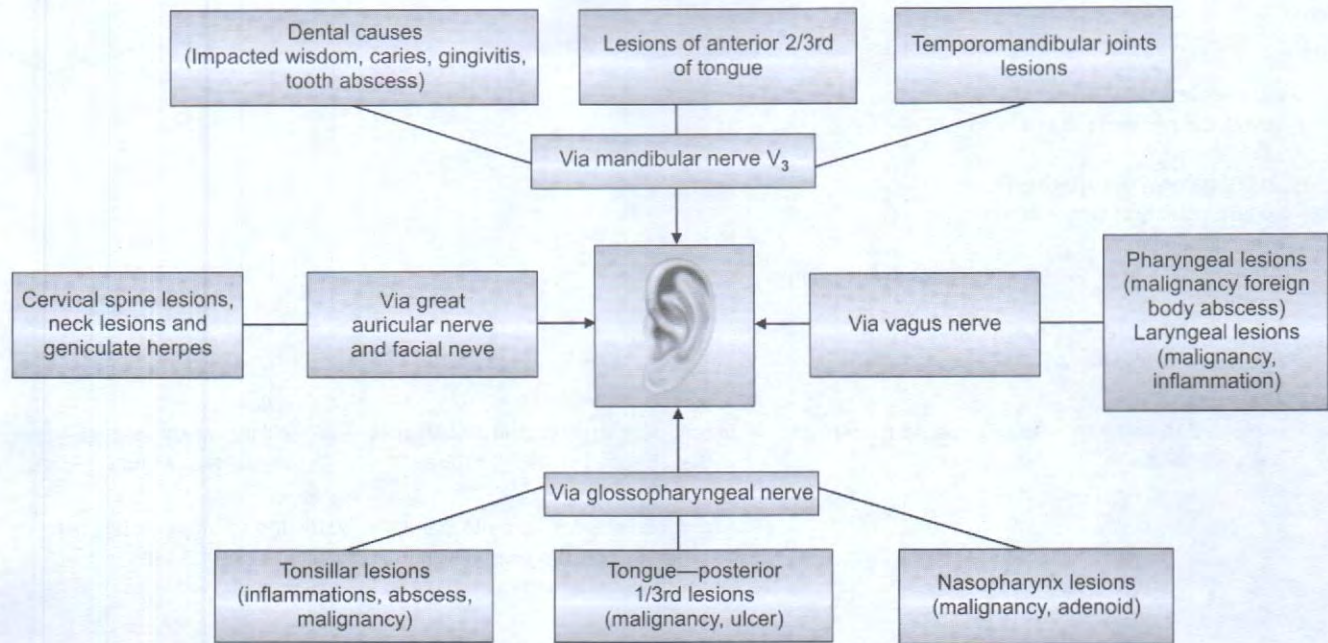


Figure 1: Causes of referred earache

Clinical Features

- Pain in ear which increases on lying down (due to increased blood supply)
- Features of underlying cause.

Treatment

Supportive	Specific
<ul style="list-style-type: none"> ♦ Analgesics to relieve pain ♦ Anesthetic ear droop to sooth earache 	<ul style="list-style-type: none"> ♦ Treatment of underlying cause

4. Eustachian tube patency tests.

- Eustachian tube is an osseocartilagenous tube connecting middle ear cavity to nasopharynx
- Patency of Eustachina tube is essential for normal functioning of ear.

Tests for Eustachian Tube Patency

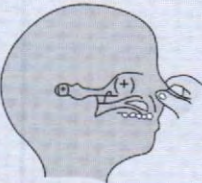
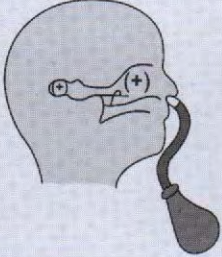
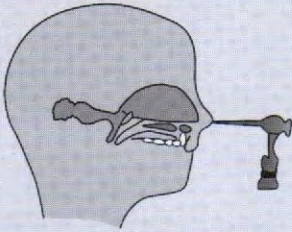
	Procedure	Result
a. Valsalva test <ul style="list-style-type: none"> – Valsalva maneuver is an Eustachian tube function test to test its patency – Also called insufflation test 	Principle <ul style="list-style-type: none"> ♦ To build positive pressure in nasopharynx so that air enters Eustachian tube Procedure <ul style="list-style-type: none"> ♦ Ask patient to pinch his nose with thumb and index finger and take a deep breath ♦ Then closing his mouth, ask him to blow air into ears 	Observation <ul style="list-style-type: none"> ♦ Observe movements of tympanic membrane using otoscope Inference <ul style="list-style-type: none"> ♦ Outward movement of tympanic membrane indicates normal functioning Eustachian tube ♦ Hissing sound indicates tympanic membrane perforation ♦ Crackling sound indicates fluid in middle ear

Figure 2: Valsalva method

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	Procedure	Result
<p><i>Disadvantages</i></p> <ul style="list-style-type: none"> – Not all can perform this test successfully <p><i>Applications</i></p> <ul style="list-style-type: none"> – To test patency of Eustachian tube – To test mobility of tympanic membrane <p><i>Contraindications</i></p> <ul style="list-style-type: none"> – Atrophic scar of tympanic membrane (which may rupture) – Infection of nose and nasopharynx (pushing of infection into middle ear) 		
<p>b. Politzer test</p> <ul style="list-style-type: none"> – Indicated in children who are unable to perform Valsalva maneuver  <p>Figure 3: Politzer's method</p> <p><i>Applications</i></p> <ul style="list-style-type: none"> – To test patency of Eustachian tube – To therapeutically ventilate middle ear 	<p><i>Principle</i></p> <ul style="list-style-type: none"> ♦ To build positive pressure in nasopharynx so that air enters Eustachian tube <p><i>Procedure</i></p> <ul style="list-style-type: none"> ♦ Introduce Politzer's bag into patient's nose on side of testing Eustachian tube ♦ Close other nose and compress Politzer's bag ♦ As patient to swallow or say "ik, ik, ik" 	<p><i>Observation</i></p> <ul style="list-style-type: none"> ♦ Auscultate patient's ear on side of testing Eustachian tube <p><i>Inference</i></p> <ul style="list-style-type: none"> ♦ Hissing sound indicates patency of Eustachian tube
<p>c. Frenzel's maneuver</p> <ul style="list-style-type: none"> – Also called nasopharyngeal pressure test 	<p><i>Procedure</i></p> <ul style="list-style-type: none"> ♦ Ask patient to move tongue up against palate against closed glottis with mouth and nose closed 	<p><i>Observation</i></p> <ul style="list-style-type: none"> ♦ Observe movement of tympanic membrane and auscultate external ear <p><i>Inference</i></p> <ul style="list-style-type: none"> ♦ Auscultation of click sound and movements of tympanic membrane indicates patent Eustachian tube
<p>d. Catheterization</p> <ul style="list-style-type: none"> – An invasive procedure of testing Eustachian tube patency  <p>Figure 4: Eustachian tube catheterization</p> <p><i>Applications</i></p> <ul style="list-style-type: none"> – To test patency of Eustachian tube 	<p><i>Principle</i></p> <ul style="list-style-type: none"> ♦ To build positive pressure in nasopharynx so that air enters Eustachian tube <p><i>Procedure</i></p> <ul style="list-style-type: none"> ♦ Anesthetize nose by topical lignocaine ♦ Introduce a Eustachian tube catheter (with bent tip) along floor of nose till it reaches nasopharynx ♦ Rotate it medially by 90° and pull it back gradually till it engages posterior border of nasal septum ♦ Then rotate it laterally by 180° so that tip of tube lies against tubal opening ♦ Connect Politzer's bag to catheter and insufflate with air 	<p><i>Observation</i></p> <ul style="list-style-type: none"> ♦ Auscultate patient's ear on side of testing Eustachian tube <p><i>Inference</i></p> <ul style="list-style-type: none"> ♦ Hissing sound indicates patency of Eustachian tube

Contd...

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	Procedure	Result
Complications <ul style="list-style-type: none"> – Scarring of tubal opening due to injury – Nasal bleeding – Transmission of nasal and nasopharyngeal infection into middle ear – Rupture of atrophic area of tympanic membrane (if too much pressure used) 		
e. Toynbee's test <ul style="list-style-type: none"> – Physiological test to test patency of Eustachian tube 	Principle <ul style="list-style-type: none"> ♦ To create negative pressure in nasopharynx so that ear from middle ear is sucked into nasopharynx Procedure <ul style="list-style-type: none"> ♦ Ask patient to swallow with nose pinched 	Observation <ul style="list-style-type: none"> ♦ Observer tympanic membrane through otoscope or microscope Inference <ul style="list-style-type: none"> ♦ Inward movement of air indicates patency of Eustachian tube
f. Tympanometry <ul style="list-style-type: none"> – Also called inflation-deflation test Advantages <ul style="list-style-type: none"> – Can be done in patients with intact and perforated tympanic membrane 	Principle <ul style="list-style-type: none"> ♦ Tests ability of Eustachian tube to balance middle ear pressure Procedure <ul style="list-style-type: none"> ♦ Create both positive and negative pressure alternatively in external ear canal while patient swallows repeatedly 	Inference <ul style="list-style-type: none"> ♦ Equilibrium of middle ear pressure to atmospheric pressure suggest normal Eustachian tubal function
g. Radiological test <ul style="list-style-type: none"> – Old method, not used nowadays Advantages <ul style="list-style-type: none"> – Also helps to analyze clearance function of Eustachian tube by estimating time taken for dye to reach nasopharynx 	Principle <ul style="list-style-type: none"> ♦ Instillation of a radiopaque dye in middle ear through pre-existing perforation and observe for tubal patency by X-rays 	
h. Saccharine or methylene blue test <ul style="list-style-type: none"> – Crude method of testing Eustachian tube patency Advantages <ul style="list-style-type: none"> – Also helps to analyze clearance function of Eustachian tube by estimating time taken for dye to reach nasopharynx 	Procedure <ul style="list-style-type: none"> ♦ Instill solution of saccharine or methylene blue in middle ear through pre-existing perforation 	<ul style="list-style-type: none"> ♦ Note time taken for sweetness or appearance of dye in nasopharynx
i. Sonotubometry <ul style="list-style-type: none"> – An under development test to test tubal patency Advantages <ul style="list-style-type: none"> – Noninvasive technique – Provides information on active tubal opening and duration for which tube remains open Disadvantages <ul style="list-style-type: none"> – Accessory sound produced in nasopharynx during swallowing may interfere with test result 	Principle <ul style="list-style-type: none"> ♦ A tone presented to ear is heard louder in presence of patent Eustachian tube when compared to obstructed Eustachian tube 	

June 2010 (RS2)

5. Rinne's test.

Refer Question No. 18 December 2007 (RS2).

6. Cerebrospinal fluid rhinorrhea.

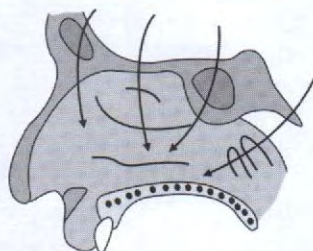
- CSF rhinorrhea is leakage of CSF through nose.

Etiology

Traumatic	Iatrogenic	Tumors	Congenital defects of skull associated with encephalocele	Spontaneous
<ul style="list-style-type: none"> ♦ Head injuries with fracture of base of skull 	<ul style="list-style-type: none"> ♦ Damage to cribriform plate during SMR operation of nasal septum ♦ Ethmoidectomy or sinus endoscopy ♦ Surgeries of sinuses ♦ Transnasal trans-sphenoidal hypophysectomy 	<ul style="list-style-type: none"> ♦ Large osteomas of frontoethmoid region ♦ Tumors of pituitary or olfactory bulb 		<ul style="list-style-type: none"> ♦ Destructive lesion resulting in destruction of floor of anterior cranial fossa ♦ Prolonged intracranial pressure due to hydrocephalus

Sites of Leakage

- Cribriform plate, ethmoid air cells/frontal sinus—anterior cranial fossa
- Sphenoid sinus—middle cranial fossa
- Temporal bone injury → middle ear → Eustachian tube → nose (otorrhorrhea).

**Figure 5:** Routes of CSF leaks**Clinical Features**

Symptoms	Signs
<ul style="list-style-type: none"> ♦ History of nasal or sinus surgery, head injury or intracranial tumor ♦ Nasal discharge <ul style="list-style-type: none"> – Thin, clear, watery – In drops or stream – On bending or straining – Uncontrollable – Cannot be sniffed back – Sweet in taste – Does not stiffen handkerchief (because does not contain mucus) 	<ul style="list-style-type: none"> ♦ Double ring sign (target sign) <ul style="list-style-type: none"> – Nasal discharge in traumatic CSF leak shows central spot of blood surrounded by clear CSF halo on a filter paper

Investigations

- Biochemical analysis of nasal discharge
 - Oxidase peroxidase paper strip test demonstrates glucose
 - Glucose content >30 mg/dL
 - B₂ transferrin is always present (CSF specific).
- Dye test
 - To locate site of CSF leak.

Procedure

- Inject 1 mL of 5% fluorescein dye or radioactive isotope intrathecally
- Placing cotton in possible sites of leakage and examine them later.

Interpretation*Stained cotton location*

- Olfactory slit
- Middle meatus
- Sphenothmoidal recess
- Inferior meatus near Eustachian tube

Site of leakage

- Cribiform plate
- Frontal or ethmoidal sinuses
- Sphenoid recess
- Temporal bone

c. CT cisternogram

- Injection of contrast material intrathecally via cisterna magna followed by CT scan.

d. Magnetic resonance imaging (MRI).

Treatment

	<i>Conservative</i>	<i>Operative</i>
Indications	♦ Early cases	♦ Persistent cases
Techniques	♦ Complete bed rest with raised head position ♦ Avoid blowing of nose, sneezing and straining ♦ Prophylactic antibiotics to prevent meningitis for 14–21 days	♦ Closure of leak by nasal endoscopic or intracranial approach

Complications

- Meningitis.

7. Rhinosporoidiosis.

- Rhinosporoidiosis is a chronic fungal granulomatous infection of nose.

Etiology

<i>Causative agent</i>	<i>Source of infection</i>	<i>Predisposing factors</i>
♦ Rhinosporidium seeberi (MC) ♦ Rhinosporidium kinealyi	♦ Contaminated pond water ♦ Dust mixed with dung of infected cattle	♦ Age: Usually in young adults ♦ Sex: Mostly in males ♦ Geography: Along coastal areas of tropical countries like India, Sri Lanka, Bangladesh, Africa and South America and in India, states like Kerala, Karnataka, Tamil Nadu, Maharashtra, Odisha ♦ Occupation: Mostly farmers

Mode of Spread

- a. DeMellow's theory of direct transmission
 - It postulates that infection always occurred as a result of direct transmission of organism when nasal mucosa came into contact with infected material while bathing in common ponds.
- b. Autoinoculation theory of Karunarathnae
 - Satellite lesions in skin and conjunctival mucosa are result of autoinoculation.
- c. Hematogenous spread
 - Rhinosporoidiosis of distant sites may occur due to hematogenous spread.

Classification

- Nasal
- Nasopharyngeal
- Mixed
- Bizzare (ocular and genital)
- Malignant rhinosporoidiosis (cutaneous).

Pathology

Gross appearance	Microscopic appearance
<ul style="list-style-type: none"> ♦ Leafy, mulberry like polypoid mass ♦ Pink to purple in color ♦ Surface studded with white gray spots especially inferior surface ♦ Very vascular and bleeds on touch 	<ul style="list-style-type: none"> ♦ Thick walled sporangia containing large number of organisms of size of erythrocytes with chitinous wall ♦ Each sporangium contains a few thousand spores which are discharged into submucosa or onto surface of mucosa ♦ Intervening tissue shows hyperplasia, focal thinning and occasional ulcerations consisting of inflammatory granulation tissues like plasma cells, lymphocytes, histiocytes, neutrophils ♦ Rhinosporidial spores stain with Sudan black, bromophenol blue, etc.

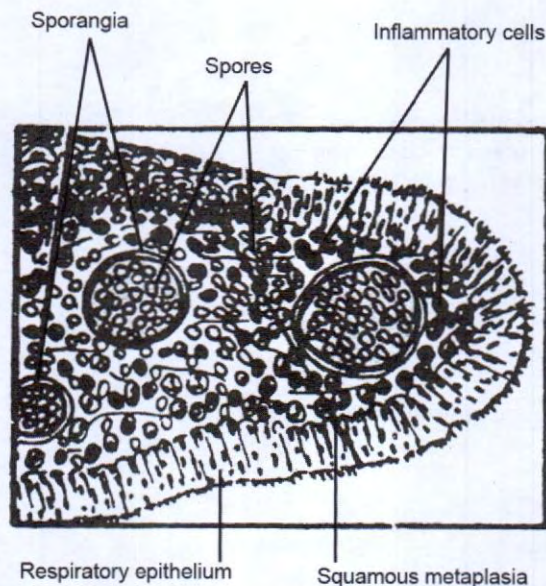


Figure 6: Rhinosporidiosis—microscopic appearance

Clinical Features

Symptoms	Signs (Anterior rhinoscopy)
<ul style="list-style-type: none"> ♦ Nasal discharge, often blood stained ♦ Epistaxis ♦ Nasal stuffiness ♦ Rhinorrhea ♦ Protruding nasal mass 	<ul style="list-style-type: none"> ♦ Leafy, strawberry like polypoid mass in nasal cavity ♦ Pink to purple in color with surface studded with white gray spots (representing fungal sporangia) ♦ Attached to nasal septum or lateral wall of nose ♦ Very vascular and bleeds on touch ♦ Painless ♦ May extend into nasopharynx or oropharynx

Spread

- Occasionally involves eyes, pharynx, trachea and genitals
- May also involve liver, spleen and skin by hematogenous spread.

Investigations

- Biopsy
 - Reveals several round or oval sporangia filled with spores seen bursting through its chitinous wall.

Treatment

<i>Conservative</i>	<i>Operative (mainstay of treatment)</i>
a. Dapsone – 100 mg/day for 6 months (tried with limited success) b. Amphotericin B – In patients with widespread disease	a. Complete excision – Using diathermy knife followed by cauterization of base – Cryosurgery – Laser surgery – Endoscopic surgery

Prognosis

- May recur even after complete excision.

8. Submucous resection (SMR) versus septoplasty.

<i>Points of difference</i>	<i>Septoplasty</i>	<i>SMR</i>
♦ Type of operation	Conservative	Radical
♦ Principle	Reconstruction/realignment	Resection
♦ Preferred in	Children	Adults
♦ Elevation of flaps	On concave side only	On both sides
♦ Cartilage	Preserved as much as possible	Removed
♦ Correction of caudal dislocation	Done	Not done
♦ Septal perforation	Rare	Higher incidence
♦ Postoperative external nasal deformity	Absent	Supratip saddling and columellar retraction
♦ Revision surgery	Easier	Difficult
♦ Combination with rhinoplasty	Done	Difficult

9. Membranous tonsillitis.

- Membranous tonsillitis is a pathological type of acute tonsillitis.

Etiopathogenesis

<i>Causative organism</i>	<i>Pathogenesis</i>	<i>Pathology</i>
♦ Hemolytic streptococci ♦ Staphylococci ♦ Pneumococci ♦ <i>H. influenzae</i>	♦ Bacterial infection of tonsil may be primary or secondary to viral infection of upper respiratory tract	♦ Membrane on tonsillar surface due to coalescing of purulent material which fills tonsillar crypts

Clinical Features

- Common in school going children.

<i>Symptoms</i>	<i>Signs</i>
♦ Sore throat ♦ Difficulty in swallowing ♦ Fever (38–40°C) with chills and rigor ♦ Earache (due to referred pain or secondary otitis media) ♦ Constitutional symptoms like headache, malaise, body aches	♦ Coated tongue ♦ Foetid breath smell ♦ Hyperemia of pillars, soft palate and uvula ♦ Whitish membrane on medial surface tonsil which could be wiped away with swab ♦ Tender and enlarged jugulodigastric nodes

Investigations

- | | |
|-------------------------|-----------------------------------|
| a. Blood investigations | ♦ Cell count reveals leukocytosis |
| b. Tonsillar swab | ♦ For culture and sensitivity |

Treatment

Supportive	Specific
	Conservative
♦ Bed rest and plenty of fluid intake	a. Antibiotics <ul style="list-style-type: none"> – To control and prevent spread of infection – Penicillin (drug of choice) or erythromycin for 7–10 days b. Analgesics <ul style="list-style-type: none"> – To relieve local pain and reduce fever

Complications

- Chronic tonsillitis due to recurrent attacks
- Peritonsillar abscess
- Parapharyngeal abscess
- Cervical abscess due to suppuration of jugulodigastric lymph nodes
- Acute otitis media
- Rheumatic fever and acute glomerulonephritis (due to group A beta hemolytic streptococci infection)
- Subacute bacterial endocarditis (due to *Streptococcus viridans* infection).

10. Sleep apnea syndrome.

- Sleep apnea is at least 30 apneic episodes each lasting for 10 seconds or more during a bout of sleep lasting for 7 hours.

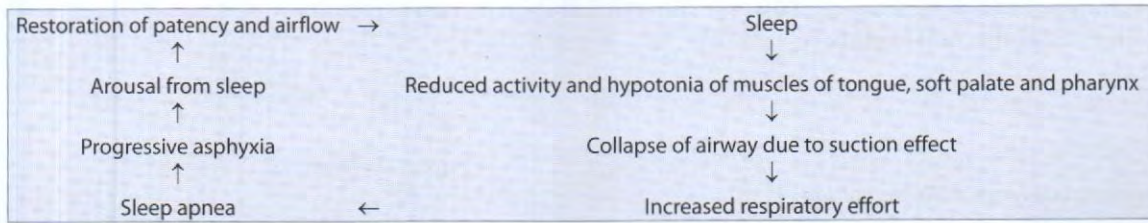
Types

Central	Obstructive	Mixed
♦ No airflow and no respiratory effort ♦ Breathing resumes again due to stimulation of respiratory centers by hypoxia ♦ Repeating cycle of breathing and apnea throughout sleep	♦ No airflow despite respiratory effort ♦ Due to upper respiratory tract obstruction ♦ Seen exclusively in males ♦ Associated with loud snoring and abnormal movements during sleep	♦ Combination of failure of central control and upper airway obstruction

Etiology

Etiology			Predisposing factors
Physiological	Anatomical	Pathological	
	♦ Macroglossia ♦ Subtle reduction in airway size ♦ Retrognathia	♦ Deviated nasal septum (DNS) ♦ Nasal polyposis ♦ Adenotonsillar hypertrophy ♦ Laryngeal edema ♦ Malignancy of upper respiratory tract ♦ Myxedema ♦ Chronic obstructive pulmonary disease (COPD)	♦ Obesity ♦ Alcohol intake ♦ Drugs

Pathogenesis



Clinical Features

- Occurs at any age but increased incidence after 40 years
- Common in males.

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Snoring (due to narrowing of airway during sleep) ♦ Nocturnal asphyxia ♦ Frequent arousal from sleep ♦ Morning headaches ♦ Day time sleepiness ♦ Nocturnal enuresis and impotence 	<ul style="list-style-type: none"> ♦ Snoring ♦ Features of airway obstruction ♦ Obstructive lesion of upper respiratory pathway

Investigations

- Polysomnography (detailed examination during sleep with monitoring of sleep stage)
- Transcutaneous monitoring of O₂ saturation during sleep
- Pulmonary function tests
- X-ray and CT of nose and pharynx to diagnose site of obstruction
- Diagnostic nasal endoscopy
- Muller's maneuver
- Multiple sleep latency test.

Treatment

Supportive	Specific	
	Conservative	Operative
<ul style="list-style-type: none"> ♦ Weight reduction ♦ Cessation of alcohol and drug ♦ Physiotherapy 	<ul style="list-style-type: none"> ♦ Continuous positive airway pressure ventilation ♦ Prosthetic devices to retain tongue in forward position 	<ol style="list-style-type: none"> Nasal or nasopharyngeal surgery <ul style="list-style-type: none"> – In cases with nasal or nasopharyngeal obstruction Adenotonsilectomy <ul style="list-style-type: none"> – In cases with obstruction due to hypertrophied tonsils or adenoids Uvulopalatopharyngoplasty (using laser or radiofrequency waves) <ul style="list-style-type: none"> – Excision of tonsils, pillars, uvula and ring of posterior part of soft palate Tracheostomy <ul style="list-style-type: none"> – In extreme cases

Complications

- Pulmonary or systemic hypertension
- Mental dysfunction and personality disorder
- Myocardial infarction, stroke
- Cardiac arrhythmias, heart failure, death.

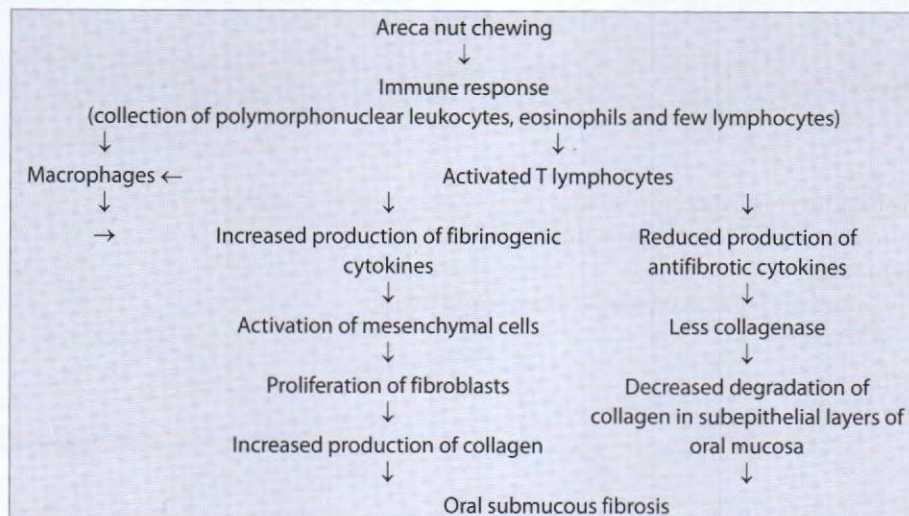
11. Oral submucous fibrosis.

- Oral submucous fibrosis is a collagen disorder affecting oral cavity and occasionally pharynx
- Also called idiopathic oral fibrosis.

Etiology

Risk factors (TAANI – Anushka Sharma's name in Rab ne mila di Jodi)	Predisposing factors
<ul style="list-style-type: none"> ♦ Tobacco chewing—major risk factor ♦ Areca nut chewing—causative agent if chewed alone, with tobacco or in form of pan ♦ Alcohol—increases risk by 2 fold ♦ Nutritional deficiency—deficiency of vitamins and micronutrients due to lesser intake of fruits and vegetables ♦ Immune response—due to cell-mediated immune reaction to arecoline in areca nuts 	<ul style="list-style-type: none"> ♦ Low socioeconomic condition ♦ Multifactorial (synergistic effect of numerous factors like tobacco chewing, alcohol)

Pathogenesis



Pathology

Initial stage	Later stage
<ul style="list-style-type: none"> ♦ Presence of polymorphonuclear leukocytes, eosinophils and few lymphocytes ♦ Fibroblastic transformation of connective tissue in lamina propria associated with epithelial atrophy 	<ul style="list-style-type: none"> ♦ Presence of lymphocytes and plasma cells ♦ Marked fibrosis

Clinical Features

- Mostly affects females aged 20–50 years.

Stages	Symptoms	Signs
Prodromal	<ul style="list-style-type: none"> ♦ Difficulty in chewing ♦ Intolerance to chilies and spicy food 	<ul style="list-style-type: none"> ♦ Patchy redness of mucosa over soft palate, faucial pillars and buccal mucosa with formation of vesicles which may burst forming superficial ulcers
Initial	<ul style="list-style-type: none"> ♦ Soreness of mouth with constant burning sensation, worsening during meals (pungent spicy food) 	<ul style="list-style-type: none"> ♦ Blanching of mucosa with loss suppleness ♦ Vertical and circular palpable fibrotic bands in affected area resulting in marble-like appearance of mucosa

Contd...

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Stages	Symptoms	Signs
	<ul style="list-style-type: none"> ♦ Recurrent ulcers in mouth (on palate and pillars) 	<ul style="list-style-type: none"> ♦ Restrictive mobility of soft palate, tongue and jaw due to fibrosis and scarring of underlying muscles ♦ Obliteration of vestibule of mouth and inability to puff out cheeks ♦ Small contracted uvula ♦ Stiff and small tongue ♦ Progressive trismus (incisor bite reduced to 2.5 cm from normal 4.5 cm) ♦ Poor orodental hygiene with carious teeth
Advanced	<ul style="list-style-type: none"> ♦ Difficulty to open mouth fully ♦ Difficulty to protrude tongue 	<ul style="list-style-type: none"> ♦ Leukoplakia ♦ Speech and hearing deficit (because of involvement of tongue and Eustachian tube)

Investigations

a. Blood picture	♦ Anemia
b. ESR	♦ Raised
c. Biopsy	<ul style="list-style-type: none"> ♦ Atrophy of epithelial layers with increased mitotic activity in basal layer ♦ Increased thickening and hyalinized collagen and fibrotic tissue in subepithelial tissue ♦ Infiltration by inflammatory cells

Differential Diagnosis

Leukoplakia	Lichen planus	Thrush
<ul style="list-style-type: none"> ♦ Thickened mucosa with cracks on patches ♦ Usually affects cheeks, lip and tongue 	<ul style="list-style-type: none"> ♦ Thin white submucosal bands ♦ Also affects skin 	<ul style="list-style-type: none"> ♦ Fungal infection with curd-like plaques

Treatment

Supportive	Specific	
	Conservative	Operative
<ul style="list-style-type: none"> ♦ Avoid irritants like areca nuts, pan, tobacco, spicy foods ♦ Vitamin A, zinc and antioxidants to treat nutritional deficiency ♦ Dental hygiene ♦ Jaw opening exercises 	<ul style="list-style-type: none"> ♦ Steroids <ul style="list-style-type: none"> – Injection of 1 mL dexamethasone (4 mg) along with 1500 IU hylase into affected area on either side twice weekly for 8–10 weeks 	<p><i>Indications</i></p> <ul style="list-style-type: none"> ♦ To relieve trismus in advanced cases <p><i>Techniques</i></p> <ul style="list-style-type: none"> ♦ Simple release of fibrosis and skin grafting ♦ Bilateral tongue flap ♦ Nasolabial flap ♦ Island palatal mucoperiosteal flap ♦ Bilateral radial forearm free flap ♦ Surgical excision and buccal fat pad graft ♦ Superficial temporal fascia flap and split skin graft ♦ Coronoidectomy and temporal muscle myotomy

Complications

- Malignant transformation (3–8%).

12. Eagle's syndrome.

- Also called styalgia, elongated styloid process.

Etiology

- Elongated styloid process
- Angulated styloid process
- Calcification of stylohyoid ligament.

Clinical Features

- May be unilateral or bilateral.

Symptoms		Signs
Classical type	Styloid process—carotid artery syndrome	
<ul style="list-style-type: none"> ♦ Dull or intermittent pain in throat and upper neck radiating to ipsilateral ear (due to glossopharyngeal neuralgia) ♦ Pain aggravated on swallowing ♦ Difficulty in swallowing ♦ Foreign body sensation in throat 	<ul style="list-style-type: none"> ♦ Parietal headache (due to disturbance in circulation) ♦ Pain along distribution of artery involved (due to irritation of nerve plexus around vessels) 	<ul style="list-style-type: none"> ♦ Tenderness in tonsillar fossa ♦ Palpable elongated styloid process in tonsillar fossa

Investigations

- X-ray (AP view with open mouth and lateral view of skull)
 - Reveals elongated styloid process.

Treatment

Conservative	Operative	
♦ Analgesics to relieve pain	Excision of styloid process	
	↓	↓
	Transoral approach (treatment of choice) <ul style="list-style-type: none"> ♦ Tonsillectomy followed by exposure of styloid process in tonsillar fossa ♦ Excise elongated portion of styloid process after elevating periosteum around it 	Cervical approach <ul style="list-style-type: none"> ♦ Incise along anterior border of sternomastoid from tip of mastoid till hyoid bone ♦ Retract anterior border and expose styloid process by deep dissection ♦ Excise elongated portion of styloid process after elevating periosteum around it

SHORT ANSWERS**13. Hennebert's sign.**

- False positive fistula test is termed Hennebert's sign.

Etiology

- Early stage of congenital syphilis
- Meniere's disease (25%).

Pathogenesis

- Stimulation of utricular macula by movement of stapes:
 - Due to hypermobile stapes footplate in congenital syphilis
 - Due to fibrous bands connecting utricular macula to stapes footplate in Meniere's disease.

14. Malignant otitis externa.

Refer Question No. 11 December 2010 (RS2).

15. Bing's test.

Refer Question No. 18 December 2007 (RS2).

16. Anterior rhinoscopy.

- Anterior rhinoscopy is method of examination of nasal cavity.

Procedure

- Ask patient to sit opposite to examiner
- Introduce fully closed nasal speculum (Thudicum or Vienna type) held in left hand
- Once in nasal cavity, fully open speculum to examine nasal cavity by focusing light at different parts of nose by tilting head of patient in various directions
- While removing speculum, keep it partially open (to avoid trapping any hair in it).

Structures seen

- Nasal septum
- Anterior part of inferior turbinate
- Inferior meatus
- Middle turbinate
- Middle meatus
- Floor of nasal cavity

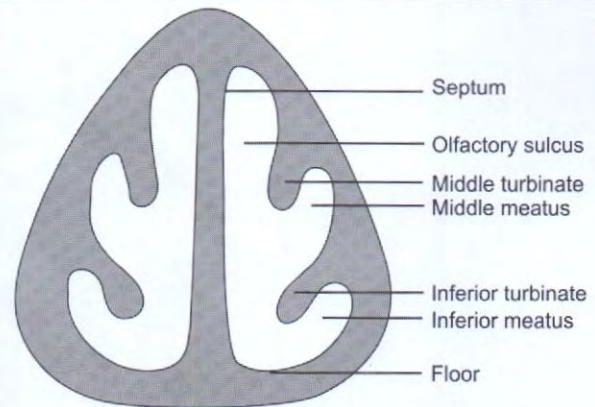


Figure 7: Anterior rhinoscopy—structures seen

17. Postnasal drip.

- Excessive nasal discharge coming into oropharynx is termed postnasal drip.

Etiology

- Allergic or infective diseases of nose and paranasal sinuses
- Adenoiditis
- Thornwaldt's disease (bursitis). (Lost 3m)

Clinical Features

Symptoms	Signs
<ul style="list-style-type: none"> Constant <u>swallowing or spitting</u> of mucus <u>Hawking</u> to clear throat Bad breath (<u>Halitosis</u>) from posterior tongue <u>Mucus feeling</u> at back of throat Broken or <u>crackling voice</u> 	<ul style="list-style-type: none"> <u>Congestion</u> of nose or paranasal sinuses <u>Sore throat</u> <u>Cobblestone appearance</u> of oropharyngeal mucosa

Treatment

Supportive	Specific
<ul style="list-style-type: none"> <u>Nasal irrigation</u> to clear nasal discharge 	<ul style="list-style-type: none"> Treatment of <u>underlying cause</u>

18. Jarjavay fracture.

- Jarjavay fracture is horizontal fracture of nasal bone involving perpendicular plate of ethmoid and septal cartilage in combination with fracture of frontal process of maxillae.

Etiology

- Lateral trauma of moderate severity (class 2).

Clinical Features

Symptoms	Signs
<ul style="list-style-type: none"> ♦ History of trauma ♦ Epistaxis ♦ Deformity of nose 	<ul style="list-style-type: none"> ♦ Tenderness ♦ Swelling ♦ Hematoma ♦ Periorbital and subconjunctival ecchymosis

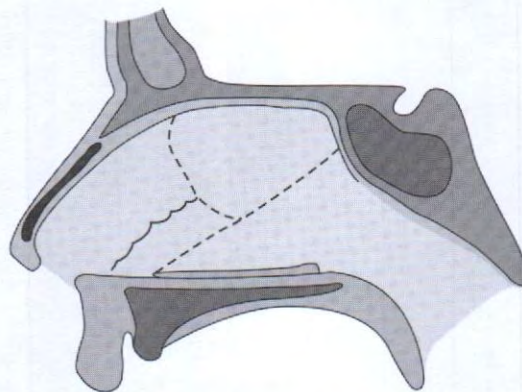


Figure 8: Jarjavay fracture

Investigations

- X-ray of nose (AP and lateral view)
 - To assess extent of fracture.

Treatment

Conservative	Operative
a. Antibiotics <ul style="list-style-type: none"> – To prevent or control infection b. Analgesics <ul style="list-style-type: none"> – To relieve pain 	a. Internal reduction <ul style="list-style-type: none"> – Reduction of fracture and excision of septal fracture and overlapping segments through a Killian incision

19. Anterior nasal packing.

Refer Question No. 1 June 2009 (RS2).

20. Reinke's edema.

- Edema of Reinke's space, i.e. subepithelial space of vocal cords
- Also called polypoid degeneration of vocal cords, bilateral diffuse polyposis of vocal cords or localized hypertrophied laryngitis.

Etiology

Etiology	Predisposing factors
<ul style="list-style-type: none"> ♦ Vocal cord abuse 	<ul style="list-style-type: none"> ♦ Heavy smoking ♦ Chronic sinusitis ♦ Laryngopharyngeal reflex ♦ Myxedema

Clinical Features

- Seen in middle age group of 40–60 years
- Common in females.

Symptoms	Signs (on indirect laryngoscopy)
<ul style="list-style-type: none"> ♦ Severe hoarseness of voice ♦ Intermittent aphonia ♦ Low pitched and rough monotonous voice (due to use of false vocal cords) 	<ul style="list-style-type: none"> ♦ Bilateral, symmetrical, spindle shaped fusiform swelling of vocal cord in membranous part ♦ Appear as translucent, grayish pink ♦ Hyperemic and hypertrophied ventricular bands

Treatment

Supportive	Specific	
	Conservative	Operative
<ul style="list-style-type: none"> ♦ Voice rest ♦ Cessation of smoking 	<ul style="list-style-type: none"> ♦ Speech therapy 	<ul style="list-style-type: none"> ♦ Decortications of vocal cords by microlaryngosurgery <ul style="list-style-type: none"> – Stripping of mucosa of vocal cords retaining enough for epithelisation – Done on one side first, followed on other side 1month later to prevent anterior glottic web formation

21. Functions of larynx.

Refer Question No. 12 December 2012 (RS2).

22. Bilateral adductor paralysis.

- Bilateral adductor paralysis is a functional disorder of speech seen as manifestation of hysteria characterized by sudden loss of voice in a normal healthy person.

Seen in	Precipitating factor
<ul style="list-style-type: none"> ♦ Emotionally sensitive personality 	<ul style="list-style-type: none"> ♦ Emotional crisis

Clinical Features

- Seen in age group of 15–30 years
- Usually affects young females.

Symptoms	Signs
<ul style="list-style-type: none"> ♦ History of emotional disturbance ♦ Sudden loss of voice or voice may be reduced to whisper ♦ No hoarseness ♦ Pain in throat ♦ Ability to whisper, cough or cry retained 	<ul style="list-style-type: none"> ♦ Vocal cords in abducted position and fail to adduct on phonation ♦ Adduction of vocal cords seen on coughing ♦ Anxious or depressed appearance of patient

Differential Diagnosis

Cerebral concussion	Aphasia
<ul style="list-style-type: none"> ♦ Similar features ♦ Rapid recovery 	<ul style="list-style-type: none"> ♦ Features of other neurological involvement

Treatment

Reassurance	Persuasion	Speech therapy	Shock therapy	Psychotherapy
<ul style="list-style-type: none"> ♦ Reassurance to patient about restoration of laryngeal function 	<ul style="list-style-type: none"> ♦ Trying to restore voice by persuading patient to cough and utilize coughing to pronounce words like "Aa" and "Ba" 	<ul style="list-style-type: none"> ♦ Very useful 	<ul style="list-style-type: none"> ♦ Faradic stimulation of larynx or intravenous injection of calcium gluconate may be tried 	<ul style="list-style-type: none"> ♦ Required in stubborn cases

Prognosis

- Good but recurrences are common.

MBBS PHASE III EXAMINATION

DECEMBER 2010

(Revised Scheme 2)

LONG ESSAYS

1. Enumerate the causes of conductive deafness in a 15 years old girl and discuss otosclerosis.

For conductive deafness.

Refer Question No. 3 December 2008 (RS2).

Otosclerosis

- Otosclerosis is a primary disease of bony labyrinth characterized by fixation of stapes to oval window, due to deposition of new bone in annular ligament of stapes
- Also called as otospongiosis or ankylosis of footplate of stapes.

Etiology

a. Age	<ul style="list-style-type: none">♦ Affects usually in 3rd decade of life♦ Rare before 10 years and 40 years of age
b. Sex	<ul style="list-style-type: none">♦ Females twice than males (especially during pregnancy)
c. Heredity (50% cases)	<ul style="list-style-type: none">♦ Autosomal dominant with incomplete penetrance and variable expressivity
d. Blood groups	<ul style="list-style-type: none">♦ In a family, affected patients share same blood group
e. Race	<ul style="list-style-type: none">♦ Whites > negroids and mongoloids♦ High incidence in southern India
f. Hormonal factors	<ul style="list-style-type: none">♦ Increased incidence in females esp. during pregnancy or menopause
g. Metabolic factors	<ul style="list-style-type: none">♦ Disturbance of calcium metabolism (deposition of new bone)
h. Associated systemic diseases	<ul style="list-style-type: none">♦ Van der Hoeve syndrome (triad of blue sclera, osteogenesis imperfecta and otosclerosis)♦ Paget's disease (a component of Osteitis deformans)

Pathology

Gross Appearance

- Chalky white, grayish or yellow otosclerotic lesion
- Sometimes red, due to increased vascularity (active and rapidly progressive focus)
- Thickened and vascular overlying mucoperiosteum.
- Type I Early focus, at least $\frac{1}{2}$ of foot plate remains thin
- Type II Fairly advanced lesion involving whole of foot plate, which can be still fractured and removed
- Type III Advanced lesion involving thickened foot plate
- Type IV Obliterative focus with bone continuous with otic capsule

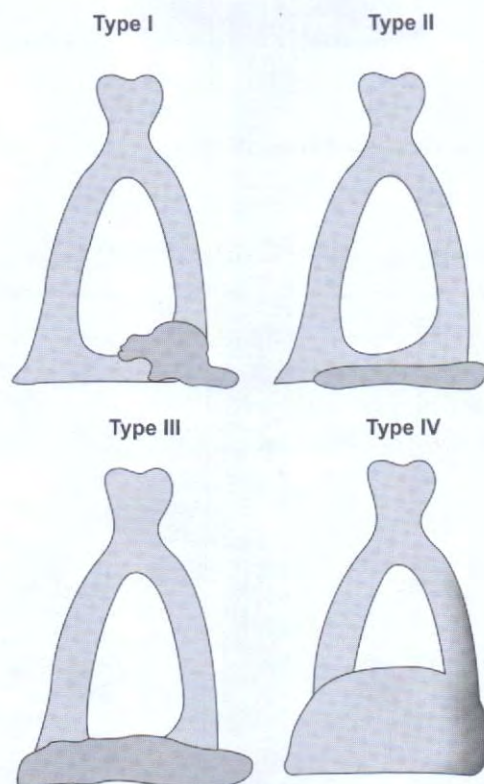


Figure 1: Otosclerosis—gross appearance

Microscopic Appearance

- Replacement of normal endochondral bone of bony labyrinth by spongy, more cellular and vascular bone
- Numerous marrow and vascular spaces with plenty of osteoblasts and osteoclasts and a lot of blue staining cement substance (blue mantles) (immature active lesion)
- Less vascularity and layer of more bone and red staining fibrillar substance than cementum (mature foci).

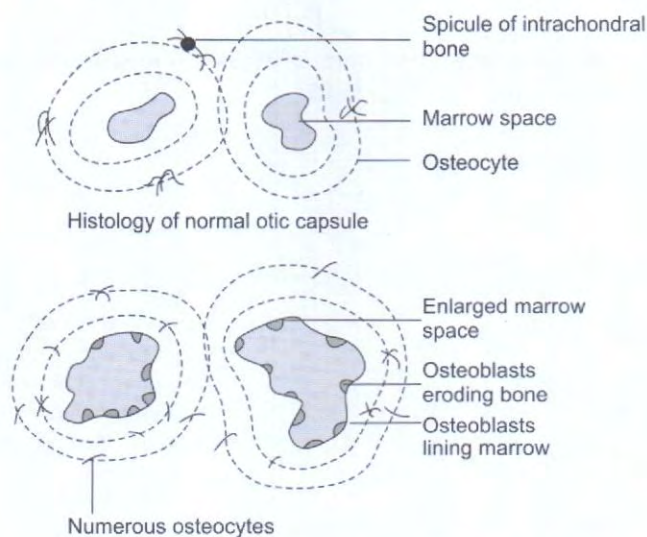


Figure 2: Otosclerosis—microscopic appearance

Types

- a. Stapedial otosclerosis
 - Most common variety
 - Characterized by stapes fixation and conductive deafness.

Types (Based on site of predilection)

Type	Site of predilection
i. Anterior focus	Fissula ante fenestram, an area 2–3 mm anterior to oval window
ii. Posterior focus	Behind oval window
iii. Circumferential type	Around margin of stapes footplate
iv. Biscuit type	In footplate with free annular ligament
v. Obliterative type	Complete obliteration of oval window niche

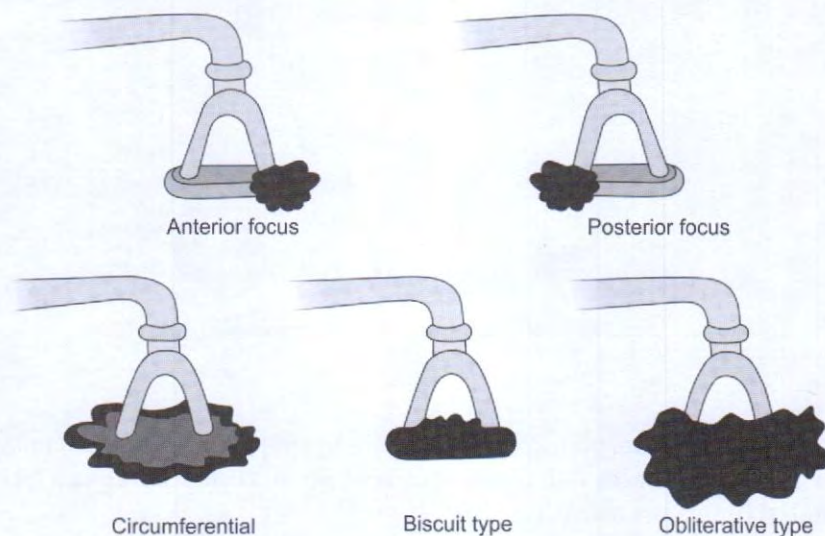


Figure 3: Stapedial otosclerosis—types

- b. Cochlear otosclerosis
 - Characterized by involvement of round window or other areas in otic capsule causing sensorineural hearing loss due to liberation of toxic materials into inner ear fluid.
- c. Histologic otosclerosis
 - Characterized by asymptomatic otosclerosis.

Clinical Features

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Hearing loss <ul style="list-style-type: none"> – Presenting complaint – Insidious in onset, starting in 20s – Often bilateral conductive type (80%) – Symmetrical – Painless and progressive ♦ Paracusis willisii <ul style="list-style-type: none"> – Ability to hear better in noisy surrounding than quite environment (because of raised voice of other people in noisy environment) 	<ul style="list-style-type: none"> ♦ Normal and mobile tympanic membrane ♦ Schwartz sign may be positive (reddish hue or flamingo pink tinge on promontory) in case of active lesion with increased vascularity ♦ Normal Eustachian tube function

Contd...

Contd...

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Tinnitus <ul style="list-style-type: none"> – Commonly seen in active lesion and cochlear otosclerosis – May be unilateral or bilateral – Usually seen in patients with early age of onset ♦ Speech <ul style="list-style-type: none"> – Monotonous, well-modulated soft speech ♦ Vertigo <ul style="list-style-type: none"> – Uncommon 	

Investigations

- a. Tuning fork tests
 - i. Rinne test
 - Negative (BC > AC) initially for 256 Hz, then 512 Hz and finally 1026 Hz (when stapes fixes completely).
 - ii. Weber test
 - Lateralized to ear with greater conductive loss.
 - iii. Gelle's test
 - Place tuning fork on mastoid process and increase pressure in external auditory canal by Seigle speculum
 - Ask patient for any change in intensity of sound after increasing pressure in external auditory canal
 - No change in otosclerotic patient due to fixed footplate of stapes (normally decrease in perceived sound).
- b. Absolute bone conduction test
 - Usually normal
 - Decreased in cochlear otosclerosis with sensorineural loss.
- c. Pure bone audiometry
 - Loss of air conduction, more for lower frequencies
 - Bilaterally symmetrical air-bone gap
 - Some cases show dip in bone conduction curve, corresponding to 5 dB loss at 1000 Hz, 10 dB loss at 1500 Hz, maximal of 15 dB loss at 2000 Hz and then reverting back to 5 dB loss at 4000 Hz producing a characteristic notch on bone conduction curve called Carhart's notch (due to increase in mass of footplate and loss of its inertial component).

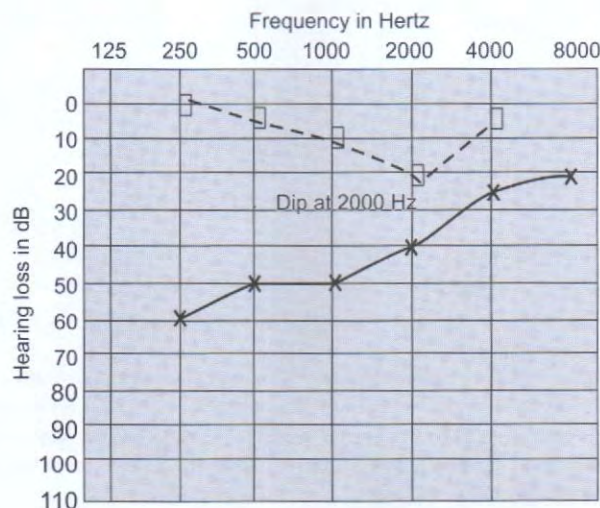


Figure 4: Carhart's notch

- d. Speech audiometry
 - Normal discrimination except in cases with cochlear involvement.

e. Tympanometry

- Normal in early cases but later shows A's type of curve (due to ossicular stiffness)
- Absent stapedial reflex when stapes becomes fixed.

Treatment*Conservative*

A. Sodium fluoride

- To hasten maturity of active focus and arrest further cochlear loss.

Mechanism of action

- Reduction of osteoclastic bone resorption and increase in osteoblastic bone formation.

Dosage

- 20 mg TID for 6 months to 1–2 years.

B. Hearing aid

- In patients refusing surgery or unfit for surgery

*Operative**Aim*

- Allow normal transmission of sound.

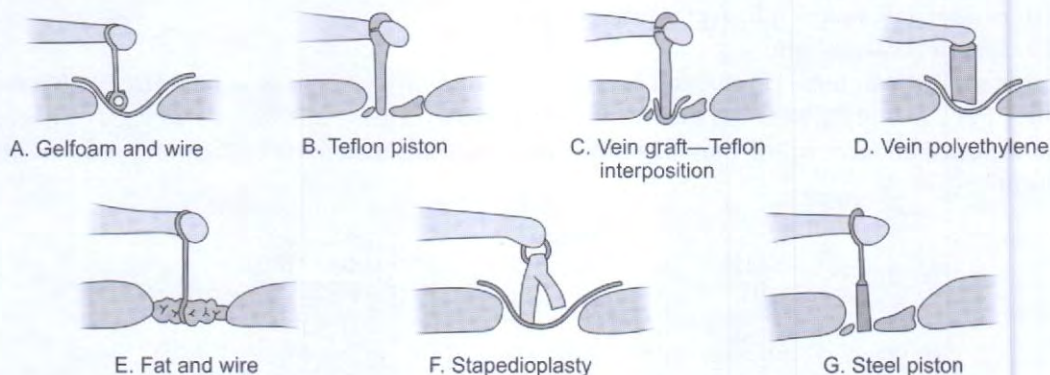
Techniques

a. Stapedectomy with prosthesis replacement

- Treatment of choice

Principle

- Removal of fixed otosclerotic stapes and insertion of a prosthesis between incus and oval window.

**Figure 5: Stapedectomy—types**

Indications	Contraindications	Ideal patient selection
<ul style="list-style-type: none"> ♦ Otosclerosis (most common) ♦ Congenital fixation of stapes ♦ Tympanosclerosis (immediate improvement in hearing) ♦ Labyrinthitis (to drain pus) 	<ul style="list-style-type: none"> ♦ Only hearing ear ♦ Associated Meniere's disease (more chances of sensorineural loss after stapedectomy) ♦ Young children (risk of displacement of prosthesis or acute otitis media) ♦ Professional athletes, high construction workers, divers and frequent air travelers (risk of postoperative vertigo, dizziness and damage hearing) ♦ Workers in noisy environment (prone for noise trauma induced sensorineural hearing loss) ♦ Extensive tympanosclerosis ♦ Malignant otosclerosis ♦ Otosclerosis with mild conductive deafness ♦ Relative contraindications like otitis externa, tympanic membrane perforation, exostosis, pregnancy 	<ul style="list-style-type: none"> ♦ Hearing threshold of 30 dB or worse ♦ Average bone-air gap at least 15 dB ♦ Rinne negative for 256 and 512 Hz ♦ Speech discrimination score of 60% or more

Anesthesia

- Local anesthesia preferred for lesser bleeding (but may be done under general anesthesia).

Position

- Supine with head turned to opposite side and pillow under ipsilateral shoulder.

Incision

- Permeatal (Rosen) incision from 6 o'clock to 12 o'clock position, 6 mm lateral to tympanic annulus at center.

Procedure

- Make incision and reflect tympanomeatal flap forward (external meatal skin and posterior ½ of eardrum) exposing middle ear

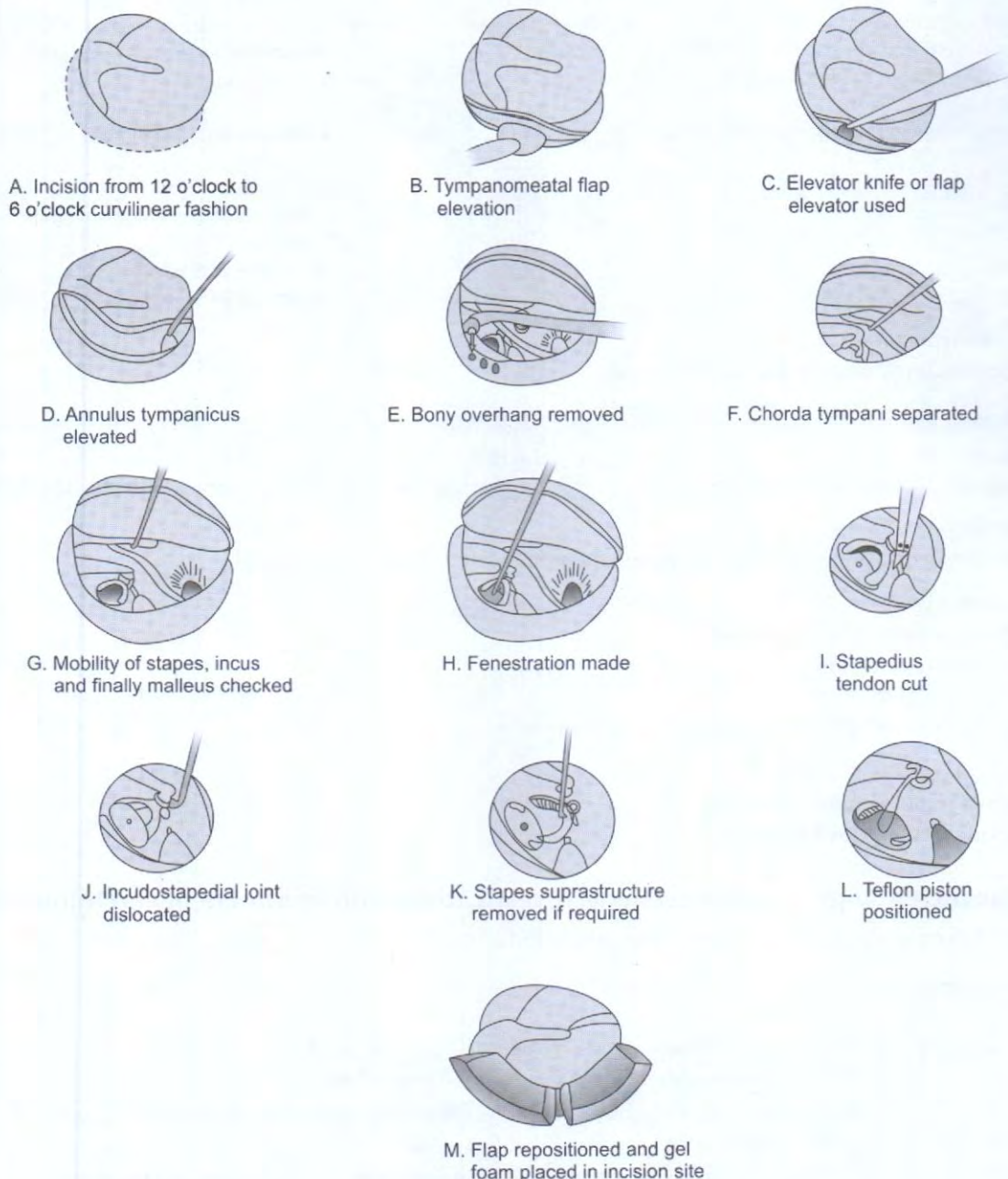


Figure 6: Stapedectomy—procedure

- Identify and preserve chorda tympani nerve
- Remove any bony overhangs of posterior canal wall
- Palpate ossicles with a probe and confirm stapedial fixation
- Cut stapedius tendon, disarticulate incudostapedial joint and break crura
- Make a hole in footplate of stapes by fine straight pick
- Take care to avoid moving of stapes more than 0.1 mm
- Hook a Teflon or stainless steel piston prosthesis around long process of incus and fit in hole made in footplate of stapes
- Put back tympanomeatal flap and pack external auditory canal with gelfoam and cotton plug.

Postoperative care	Prognosis	Complications
<ul style="list-style-type: none"> ♦ Antibiotics and analgesics ♦ Labyrinthine sedatives (to control giddiness) ♦ Decongestants (to decongest Eustachian tube and middle ear) ♦ Avoid swimming and flying for 3–4 weeks 	<ul style="list-style-type: none"> ♦ Almost normal hearing in 98% of cases 	<ul style="list-style-type: none"> ♦ Injury to chorda tympani and facial nerve ♦ Tympanic membrane perforation ♦ Hemotympanum ♦ Perilymph fistula ♦ Sensorineural hearing loss ♦ Labyrinthitis ♦ Acute otitis media ♦ Necrosis of long process of incus ♦ Slipping of prosthesis ♦ Postoperative granuloma ♦ Subluxation of footplate

- b. Laser stapedectomy
- Stapedectomy using argon or KTP lasers.

Advantages	Disadvantages
<ul style="list-style-type: none"> ♦ Bloodless fenestra ♦ Reduced risk of subluxation of footplate 	<ul style="list-style-type: none"> ♦ High cost ♦ Vestibular symptoms due to increased perilymph temperature

- c. Fenestration operation
- Involves bypassing stapes by making an opening in lateral semicircular canal.

Disadvantages

- Produces open mastoid cavity
- Residual hearing loss.

- d. Mobilization of stapes
- Mobilization of ankylosed stapes at operation.

Disadvantages

- Partial improvement in hearing
- Frequent reankylosis of stapes.

2. Describe the etiology, symptoms, signs, investigations and treatment of carcinoma larynx.

- Malignant laryngeal carcinoma are quite common in India.

Etiology ("TARGET")

a. Tobacco smoking	♦ Carcinogens like benzopyrene and other hydrocarbons in tobacco
b. Alcohol	♦ Acts synergistically with smoking increasing risk by 15 fold
c. Radiation	♦ Induction of carcinoma during radiation to neck for benign lesion or laryngeal papilloma
d. Genetic	♦ Familial tendency exhibited by laryngeal carcinoma
e. Environmental	♦ Occupation exposure to asbestos, mustard gas and other chemical or petroleum products
f. Tumor	♦ Solitary papilloma, leukoplakia, erythroplakia may turn malignant



Figure 7: Laryngeal carcinoma—etiology

Classification (AJCC Classification, 2002)

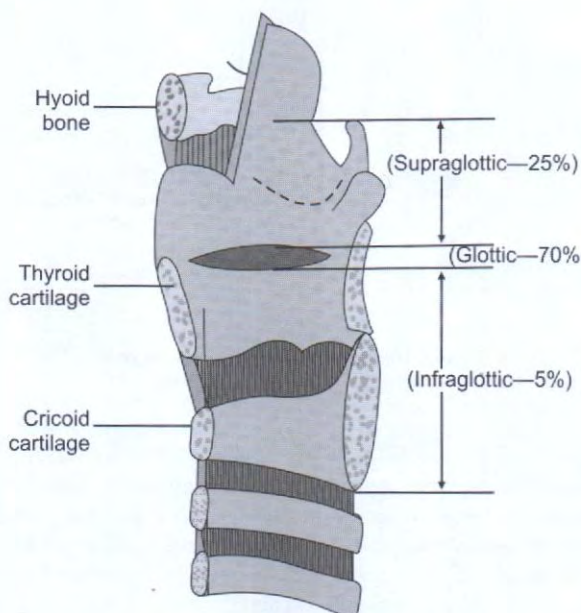


Figure 8: Laryngeal carcinoma—classification

<i>Supraglottic carcinoma (25%)</i>	<i>Glottic carcinoma (most common – 70%)</i>	<i>Subglottic carcinoma (rare – 5%)</i>
<ul style="list-style-type: none"> ♦ Suprahypoid epiglottis (both lingual and laryngeal surfaces) ♦ Infrahypoid epiglottis ♦ Ventricular bands (false cords) ♦ Aryepiglottic folds (laryngeal aspect) ♦ Arytenoids 	<ul style="list-style-type: none"> ♦ True vocal cords including anterior and posterior commissure 	<ul style="list-style-type: none"> ♦ Subglottis up to lower border of cricoids cartilage

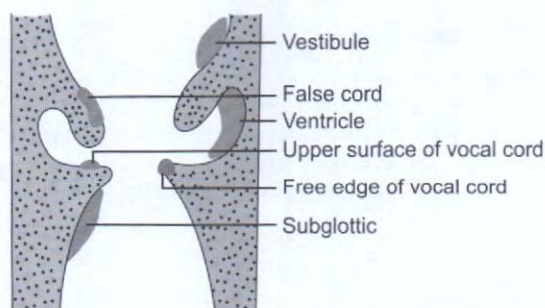
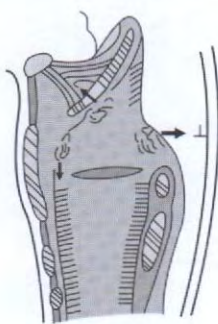


Figure 9: Laryngeal carcinoma—common sites

Pathology

Gross appearance	Microscopic examination
<ul style="list-style-type: none"> ◆ Proliferative (exophytic) usually well differentiated ◆ Ulcerative (endophytic) usually poorly differentiated ◆ Ulceroproliferative 	<ul style="list-style-type: none"> ◆ Most of lesions are squamous cell carcinoma (90–95%) with various grades of differentiation from well differentiated cordal lesions to anaplastic supraglottic lesions ◆ Rest (5–10%) include verrucous carcinoma, spindle cell carcinoma, malignant salivary gland tumors and sarcomas

Spread



Spread of supraglottic cancer
 1. From marginal region into pyriform sinus/postcricoid region
 2. Into pre-epiglottic space
 3. Inferiorly into subglottic space

Figure 10: Laryngeal carcinoma—spread

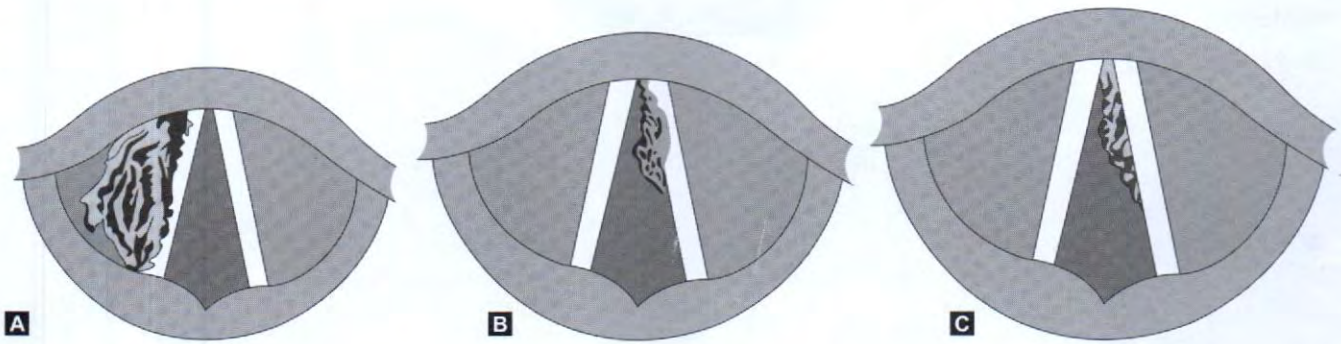
	Supraglottic cancer	Glottic cancer	Subglottic cancer
Direct spread	Adjoining areas like vallecula, base of tongue and pyriform fossa or extend into pre-epiglottic space and penetrate thyroid cartilage	Anterior commissure and then to opposite cord (anteriorly), vocal process and arytenoids region (posteriorly), ventricle and false cord (superiorly) and subglottic region (inferiorly)	Around anterior wall to opposite side or trachea and later to vocal cords, cricothyroid membrane, thyroid gland and ribbon muscles of neck
Lymphatic spread	Upper and middle jugular nodes	Does not occur (due to absence of lymphatics in cord) but may occurs if lesion spread beyond membranous cord	Prelaryngeal, pretracheal, paratracheal and lower jugular nodes

Clinical Features

- Seen mostly in adults above 40 years
- Males are 10 times more prone than females.

Symptoms

Supraglottic carcinoma	Glottic carcinoma	Subglottic carcinoma
<ul style="list-style-type: none"> ◆ Muffled voice initially becoming hoarse later at advanced stage ◆ Foreign body sensation in throat ◆ Dysphagia ◆ Throat pain ◆ Referred pain in neck and ear ◆ Lymph node mass in neck ◆ Weight loss ◆ Laryngeal obstruction ◆ Halitosis 	<ul style="list-style-type: none"> ◆ Persistent hoarseness of voice (early sign) ◆ Cough due to aspiration ◆ Stridor (due to increase in size of growth with accompanying edema) ◆ Laryngeal obstruction (due fixation of cord) 	<ul style="list-style-type: none"> ◆ Cough ◆ Hemoptysis ◆ Stridor ◆ Laryngeal obstruction ◆ Hoarseness of voice (late symptom)



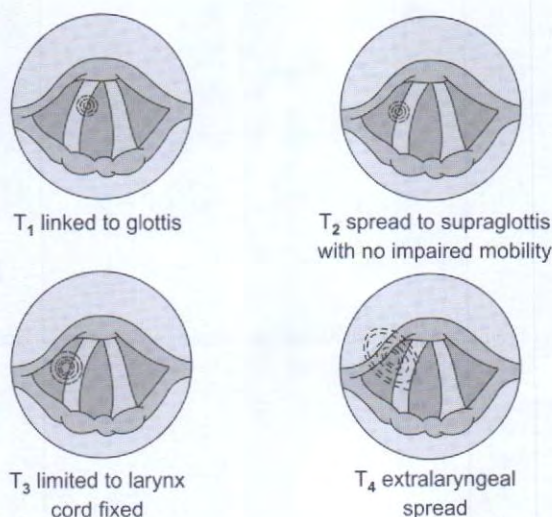
Figures 11A to C: (A) Supraglottic carcinoma; (B) Glottic carcinoma; (C) Subglottic carcinoma

Signs

- a. Tumor mass
 - Proliferative growth for suprahoid epiglottic lesion
 - Ulcerative lesion for infrahoid epiglottic lesions
 - Raised nodule or ulcer or thickening at upper surface of vocal cords in anterior two third for glottic cancer
 - Granular tissue appearance of anterior commissure cancer
 - Raised submucosal nodules involving anterior half for subglottic cancer.
- b. Vocal cord mobility
 - Impairment of vocal cord mobility due to deeper infiltration into thyroarytenoid muscle, cricoarytenoid muscle, cricoarytenoid joint or invasion of recurrent laryngeal nerve.
- c. Neck swelling
 - Midline neck swelling due spread of growth in anterior commissure and subglottic region through cricothyroid membrane.

Investigations

- a. Radiography
 - X-ray (lateral view) neck to assess extent of lesion, involvement of pre-epiglottic space or destruction of thyroid cartilage
 - X-ray chest for co-existent lung disease, pulmonary metastasis or mediastinal nodes
 - Contrast laryngograms using radiopaque dye (dionosil) to outline surface extent of tumor
 - CT scan to determine extent of tumor; invasion of pre-epiglottic or paraepiglottic space, destruction of cartilage and lymph node involvement.
- b. Diagnostic laryngoscopy
 - Direct laryngoscopy to visualize hidden areas of larynx and assess extent of disease
 - Microlaryngoscopy for small lesion of vocal cords and biopsy.
- c. Supravital staining and biopsy
 - Staining of laryngeal lesions with toluidine blue and biopsy of stained areas.

TNM Staging**Figure 12:** Laryngeal carcinoma—tumor staging

Stage	Tumor (T)			Nodal involvement (N)	Metastasis (M)
	Supraglottic	Glottic	Subglottic		
X	—	—	—	Cannot be assessed	Cannot be assessed
0	—	—	—	No metastasis	No distant metastasis
1a	Limited to one subsite with normal cord mobility	Limited on one cord with normal mobility	Limited to subglottis	Single ipsilateral node, 3 cm or less	Distant metastasis
1b		Involves both cords with normal mobility			
2a	Invasion of adjacent subsites without fixation	Invasion of supraglottis and/or subglottis with or without impaired cord mobility	Invasion of cords with or without impaired cord mobility	Single ipsilateral node, 3–6 cm	--
2b				Multiple ipsilateral nodes, <6 cm	
2c				Bilateral nodes, <6 cm	
3	Limited to larynx with cord fixation and/or invades either postcricoid area, pre-epiglottic tissue, paraepiglottic space and/or thyroid cartilage invasion	Limited to larynx with cord fixation and/or invades paraglottic space and/or minor thyroid	Limited to larynx with cord fixation	Nodes >6cm	--
4a	Invades through thyroid/cricoid cartilage and/or invades tissues beyond larynx (trachea, deep extrinsic tongue muscles, strap muscles, thyroid or esophagus)			--	--
4b	Invades prevertebral space, mediastinal structures or encases carotid artery				

Grading

Grade	Tumor (T)	Nodal involvement (N)	Metastasis (M)
0	is	0	0
I	1	0	0
II	2	0	0
III	3	0	0

Contd...

Contd...

Grade	Tumor (T)	Nodal involvement (N)	Metastasis (M)
	1/2/3	1	0
IVA	4a	0	0
	4b	1	0
	1/2/3/4a	2	0
IVB	4b	0/1/2/3	0
	1/2/3/4	3	0
IVC	1/2/3/4	0/1/2/3	1

Treatment**Conservative**

	Chemotherapy	Radiotherapy
Indications	<ul style="list-style-type: none"> ♦ Palliative (alone) ♦ Neoadjuvant (following radiotherapy or surgery) ♦ Concomitant (simultaneously with radiotherapy) 	<ul style="list-style-type: none"> ♦ Curative (early stages) ♦ Palliative (advanced stage) ♦ Neoadjuvant (following surgery) ♦ Anaplastic growths
Modalities		i. Curative – 6500 grays in 30 fractions over 5–6 weeks ii. Palliative – Lesser dosage

Operative**a. Laryngectomy**

	Conservative	Total	Near total (partial)
Indications	♦ Early stages	<ul style="list-style-type: none"> ♦ Lesions with cord fixation and beyond (T3 and T4) ♦ Invasion of thyroid or cricoids cartilage ♦ Bilateral arytenoid cartilage involvement ♦ Lesions of posterior commissure ♦ Failure of radiotherapy or conservative surgery ♦ Transglottic cancer 	
Contraindications		♦ Distant metastasis	
Procedure	♦ Excision of vocal cords after splitting larynx (cordectomy via laryngofissure)	<ul style="list-style-type: none"> ♦ Involves excision of entire larynx including hyoid bone, pre-epiglottic space, strap muscles and 1 or more tracheal rings followed by pharyngeal wall repair and suturing of lower tracheal stump to skin for breathing ♦ Partial pharyngectomy may be done if paraglottic space involved 	<ul style="list-style-type: none"> ♦ Laryngectomy with leaving behind one functioning arytenoids and healthy subglottic mucosa which is used to reconstruct dynamic shunt connecting trachea to produce aspiration free voice ♦ Excision of vocal cords and anterior commissure region (partial frontal laryngectomy)

b. Endoscopic laser excision

- In early stages using CO₂ laser

c. Neck dissection (Modified/functional/selective)

- To tackle lymph node and lymph bearing structures.

Treatment of Choice

<i>Stage I and II (organ preservation techniques)</i>	<i>Stage III and IV (combined modality)</i>
<ul style="list-style-type: none"> ♦ Radiotherapy (treatment of choice) ♦ Excision by laryngofissure technique ♦ Conservative laryngectomy ♦ Laser excision 	<ul style="list-style-type: none"> ♦ Total laryngectomy with neck dissection followed by radical radiotherapy (treatment of choice) ♦ Radical radiotherapy followed by surgery for salvage

SHORT ESSAYS**3. Fractures of middle third of face.**

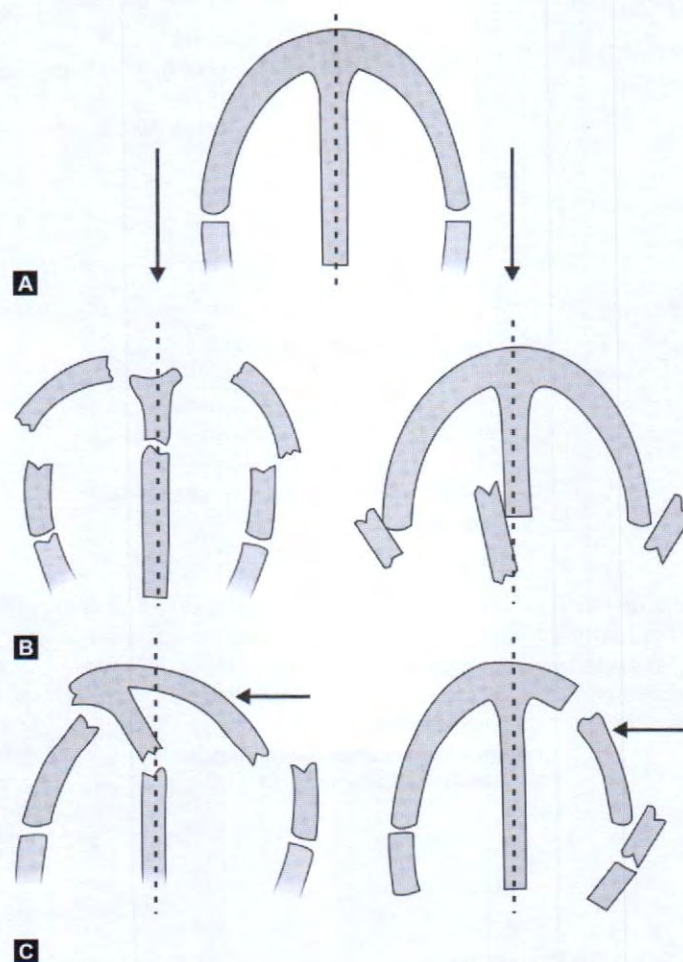
- Injuries to face occur due to assaults or accidents (industrial or transportation).

A. Nasal bone fractures

- Fractures of nasal bones are most common due to projection of nose from face.

Etiology

- Direct trauma to nose
- Associated with head injury.



Figures 13A to C: Nasal bone fracture—types

Types

<i>Depending upon direction of impact</i>		<i>Depending upon amount of force</i>	<i>Depending upon overlying soft tissue injury</i>
↓	↓		
Depressed (due to frontal blow)	Angulated (due to lateral blow)		
<ul style="list-style-type: none"> ♦ Severe frontal blow causes open book fracture (collapse of nasal septum and splaying of nasal bone) ♦ Even greater force causes comminution of nasal bones sometimes even flattening and widening nasal dorsum 	<ul style="list-style-type: none"> ♦ Unilateral depression of nasal bone on same side ♦ Fracture of both nasal bones and septum with deviation of nasal bridge 	<ul style="list-style-type: none"> ♦ Undisplaced (green stick) ♦ Displaced 	<ul style="list-style-type: none"> ♦ Open ♦ Closed

Clinical Features

<i>Symptoms</i>	<i>Signs</i>
<ul style="list-style-type: none"> ♦ History of trauma ♦ Swelling of nose ♦ Pain ♦ Nasal obstruction ♦ Epistaxis ♦ Black eye 	<ul style="list-style-type: none"> ♦ Periorbital ecchymosis ♦ Tenderness ♦ Septal hematoma ♦ Crepitus ♦ Mobility of fractured fragments ♦ Nasal deformity (depressed or deviated)

Investigations

- X-rays
 - Water's view, right and left lateral view and occlusal view.

Treatment

<i>Conservative</i>	<i>Operative</i>
<i>Indications</i> <ul style="list-style-type: none"> ♦ Simple fractures without displacement <i>Components</i> <ul style="list-style-type: none"> ♦ Antibiotics to prevent infection ♦ Anti-inflammatory drugs to reduce swelling and pain 	<i>Timing</i> <ul style="list-style-type: none"> ♦ Immediately if patient present early ♦ After 5–7 days (till edema subsides) but before 2 weeks (by when fracture heals) if patient present late <i>Techniques</i> <ol style="list-style-type: none"> Closed reduction <ul style="list-style-type: none"> – Depressed fracture reduced by straight blunt elevator guided by digital manipulation from outside – Laterally displaced fracture reduced by firm digital pressure in opposite direction – Septal fractures reduced by Asche's forces – Impacted fragments disimpacted with Walsham or Asche's forceps before realignment Open reduction <ul style="list-style-type: none"> – Indicated when closed reduction fails Rhinoplasty or septorhinoplasty <ul style="list-style-type: none"> – Indicated in cases of healing in deformed position

B. Naso-orbital fractures

- Fracture of facial bones.

Etiology

- Direct blow on nasion.

Bones fractured

- Nasal bones
- Paranasal sinuses
- Perpendicular plate of ethmoid
- Medial orbital wall
- Cribriform plate.

Clinical features

Symptoms	Signs
<ul style="list-style-type: none"> ♦ History of trauma ♦ Pain ♦ Deformity 	<ul style="list-style-type: none"> ♦ Pug nose (depressed bridge of nose with tip turned up) ♦ Periorbital ecchymoses ♦ Orbital hematoma ♦ CSF leakage (due to fracture of cribriform plate and dura) ♦ Displacement of eyeball ♦ Telecanthus (due to lateral displacement of medial orbital wall)

Investigations

- X-ray (Water's and Caldwell's view)
 - Shows fracture segments.
- CT scan
 - Investigation of choice
 - Better visualization of fracture fragments.

*Treatment**Operative*

Closed reduction	Open reduction
<ul style="list-style-type: none"> ♦ Indicated in uncomplicated cases ♦ Reduction of fracture using Asche's forceps and stabilization using wire passed through fractured bone fragments and septum 	<ul style="list-style-type: none"> ♦ Indicated in cases of extensive comminution of nasal and orbital bones or those complicated by other injuries to lacrimal apparatus, medial canthal ligament, frontal sinus, etc. ♦ Fracture site is exposed with a H shaped incision and fractures are reduced under vision along with reconstruction of nasal counter

C. Fracture of zygoma (Tripod fractures)

- Second most common fracture of face
- Called tripod fracture due to three sites of fractures namely zygomatico-maxillary, fronto-zygomatic and zygomatic arch.

Etiology

- Due to application of force to malar bone or zygomatic arch from lateral aspect leading to displacement of fracture segments backwards and downwards into antrum.

Clinical features

Symptoms	Signs
<ul style="list-style-type: none"> ♦ History of trauma ♦ Pain ♦ Facial swelling ♦ Bleeding from nose ♦ Trismus (due to entrapment of temporalis or associated fracture of coronoid process of mandible) 	<ul style="list-style-type: none"> ♦ Flattening of malar eminence immediately after fracture (masked later by facial swelling) ♦ Periorbital ecchymosis ♦ Step deformity of infraorbital rim ♦ Paraesthesia of cheek due to infraorbital nerve entrapment ♦ Narrow palpebral fissure (if lateral canthal ligament pulled inferolaterally) ♦ Restricted ocular movements (due to displacement of lateral palpebral ligament) ♦ Periorbital emphysema (due to escape of air from maxillary sinus)

Investigations

- X-ray
 - PNS (Water's and Caldwell's view)
 - * Shows fracture segments and clouding of maxillary sinus presence of blood.
 - Base of skull (Hirtz view)
 - * Show fracture of zygomatic arch.
- CT scan
 - Investigation of choice
 - Better visualization of fracture fragments with respect to posterior wall of maxilla
 - Helps diagnose comminution with depression of orbital floor and herniation of orbital contents.

*Treatment**Operative*

- Open reduction and internal fixation by wiring or plating
 - Indicated only in displaced fractures
 - Fracture is exposed at frontozygomatic suture through lateral brow incision and reduced by passing an elevator behind zygoma
 - Wire fixation is done at frontozygomatic suture and infraorbital margin.

D. Fractures of zygomatic arch

- Direct trauma to lateral part of middle third of face results in fracture of zygomatic arch.

Etiology

- Direct trauma over zygomatic arch resulting in formation of two fragments with three fracture lines (1 each at ends of fracture fragments and 1 in center of arch).

Clinical features

Symptoms	Signs
<ul style="list-style-type: none"> ♦ History of trauma ♦ Pain, aggravated on talking and chewing ♦ Deformity 	<ul style="list-style-type: none"> ♦ Loss of facial counter (due to depression in area of zygomatic arch) ♦ Limitation of movements of mandible (due to impingement of fragments on condyle or coronoid process)

Investigations

- X-ray (Submentovertical and Water's view)
 - Shows fracture segments.
- CT scan
 - Investigation of choice
 - Better visualization of fracture fragments.

*Treatment**Operative*

- Open reduction
 - Fracture site is exposed with a vertical incision in hair-bearing area above or front of ear, cutting through temporal fascia
 - Fracture is reduced after passing an elevator deep to temporal fascia.

E. Fractures of orbital floor

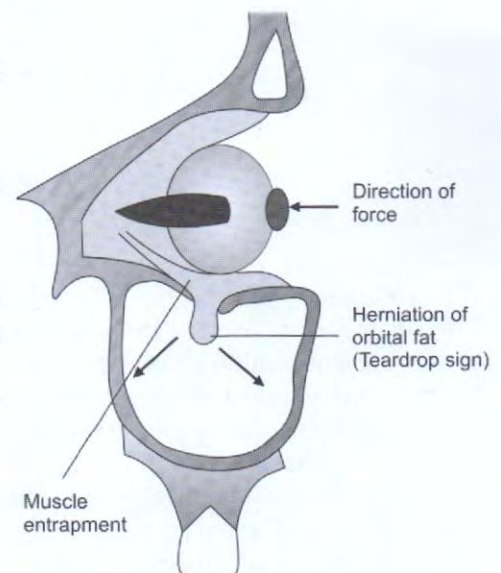
- Blow-out fractures are isolated comminuted fractures mainly involving orbital floor and medial wall.

Etiology

- Trauma to orbit by relatively large (>5 cm diameter), often rounded object (tennis ball, human fist, etc.).

Mechanism of injury

- Indirect pressure on orbital wall → backward displacement of eye → increase in intraorbital pressure → fracture of orbit at weakest point (orbital floor and medial wall) (hydraulic theory)
- Posterior transmission of compressive force at inferior orbital rim → buckling of orbital floor (buckling theory).

**Figure 14:** Blow-out fracture—mechanism

Types

Pure blow-out fractures	Impure blow-out fractures
No involvement of orbital rim	Associated with fracture of middle 1/3rd of facial skeleton

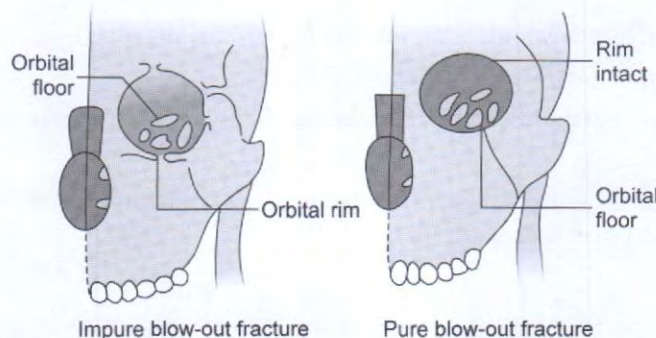


Figure 15: Blow-out fracture—types

Clinical features

Immediate	After 10 days (as oedema subsides)
<ul style="list-style-type: none"> ♦ Periorbital edema and blood extravasation in and around orbit ♦ Emphysema of eyelids (frequent with medial wall fracture) ♦ Paraesthesia and anaesthesia in distribution of infraorbital nerve (lower lid, cheek, side of nose, upper lip and upper teeth) ♦ Ipsilateral epistaxis due to bleeding from maxillary sinus into nose ♦ Proptosis of variable degree ♦ Severe ocular damage – rare 	<ul style="list-style-type: none"> ♦ Enophthalmos and mechanical ptosis because of <ul style="list-style-type: none"> – Escape of orbital fat into maxillary sinus – Backward traction on globe by entrapped inferior rectus – Enlargement of orbital cavity from displacement of fragments ♦ Double diplopia (both in up and down gaze) due to entrapment of inferior rectus and inferior oblique

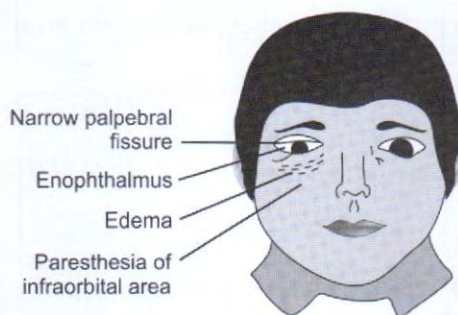


Figure 16: Blow-out fracture—clinical features

Investigations

- Plain X-rays—Water's view (nose-chin view)
 - Fragmentation and irregularities of orbital floor
 - Depression of bony fragments
 - Hanging drop opacity of superior maxillary antrum from orbital contents herniating through floor.
- CT scan and MRI
 - Useful in evaluating extent of fracture.
- Forced duction test
 - Positive, i.e. limitation of passive movements of eye in direction of deficient gaze.

Treatment

Conservative	Operative
Indications <ul style="list-style-type: none"> ♦ Small cracks or minor fractures 	Indications <ul style="list-style-type: none"> ♦ Unresolving diplopia ♦ Large herniation of tissues into antrum ♦ Incarceration of tissues in fracture ♦ Enophthalmos greater than 3 mm Timing <ul style="list-style-type: none"> ♦ 10–14 days after injury Principle <ul style="list-style-type: none"> ♦ Restore continuity of orbital floor with or without implants Technique <ul style="list-style-type: none"> ♦ Approach through lower lid ♦ Elevate periorbita from orbital floor ♦ Release entrapped inferior rectus and orbital tissue ♦ Bridge fracture opening with bone graft or silicone rubber

F. Fractures of maxilla (Le Fort fractures)

- Fractures of maxilla are called Le Fort fractures because they were classified by Le Fort of Paris in 1901.

Classification

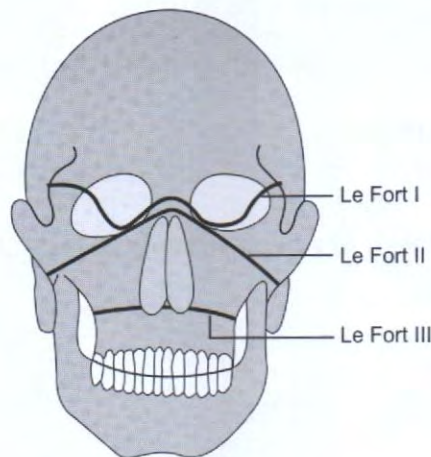


Figure 17: Le Fort fracture—classification

Le Fort I (transverse)—Guerin's fracture	Le Fort II (pyramidal)	Le Fort III (craniofacial dysfunction)
<ul style="list-style-type: none"> ♦ Fracture line passes above floor of maxillary antrum involving alveolar processes, palate and fracturing lower part of pterygoid plates 	<ul style="list-style-type: none"> ♦ Fracture involves middle 1/3rd of face, starting at midpart of nasal bone and extends laterally through pterygoid process in middle 1/3rd ♦ Fracture line passes through root of nose, lacrimal bone, floor of orbit, upper part of maxillary sinus and pterygoid plates 	<ul style="list-style-type: none"> ♦ Complete disruption of attachment of facial skeleton to cranium ♦ Fracture line passes through root of nose, ethmoidal junction, superior orbital fissure, lateral wall of orbit, frontozygomatic and temporozygomatic sutures and upper part of pterygoid plates

Clinical features (depending upon severity of trauma)

Symptoms	Signs
<ul style="list-style-type: none"> ♦ History of trauma ♦ Soft tissue swelling around nose and maxilla ♦ Nasal obstruction 	<ul style="list-style-type: none"> ♦ Collapse of nasal bridge ♦ Malocclusion of jaw ♦ Crepitus

Contd...

Contd...

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Epistaxis ♦ Paresthesia over cheek due to injury to infraorbital nerve (type 2) ♦ CSF rhinorrhea (types 2 and 3) ♦ Diplopia (types 2 and 3) ♦ Elongated face (types 2 and 3) 	<ul style="list-style-type: none"> ♦ Mobility of maxilla ♦ Step deformity <ul style="list-style-type: none"> – Pyriform aperture and palatal region (type 1) – At orbital rim and nasal bone (type 2) – At nasal bones (type 3) ♦ Dish face deformity due to flattening of face (types 2 and 3)

Investigations

- X-ray
 - Water's view, posteroanterior view, lateral view.
- CT scan

Treatment

Supportive	Specific
<ul style="list-style-type: none"> ♦ Restore airway ♦ Control hemorrhage from maxillary artery or its branches 	<p><i>Principle</i></p> <ul style="list-style-type: none"> ♦ Proper reduction of displaced fragments and maintaining it for useful bite of teeth <p><i>Techniques</i></p> <ul style="list-style-type: none"> ♦ Interdental wiring ♦ Intermaxillary wiring using arch bars ♦ Open reduction and interosseous wiring ♦ Wire slings from frontal bone, zygoma or infraorbital rim to teeth or arch bars

4. Obstructive sleep apnea syndrome.

Refer Question No. 10 June 2010 (RS2).

5. Clinical features and management of nasopharyngeal angiofibroma.

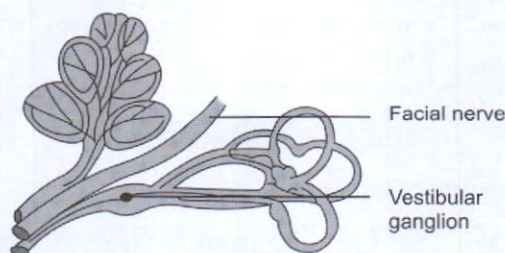
Refer Question No. 1 December 2009 (RS2).

6. Vestibular neuronitis.

- Vestibular neuronitis is peripheral vestibular disorder.

Etiology

- Idiopathic
- Viral infection affecting vestibular nerve ganglion.

**Figure 18:** Site of lesion in vestibular neuronitis**Pathophysiology**

- Self-limiting inflammatory process involving vestibular nerve.

Clinical Features

- Usually occurs between age of 30 to 50 years
- Both sexes affected equally.

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Giddiness <ul style="list-style-type: none"> – Strikes like bolt from blue and lasts few days to 2–3 weeks – Persistent and frequent in beginning and becomes paroxysmal – Spontaneously exacerbated by head movements – Usually does recur but sometimes may recur on few occasions and each successive attack weaker than former – Usually follows upper respiratory tract infection ♦ Nausea and vomiting (due to vestibular disturbance) ♦ Absence of deafness and tinnitus 	<ul style="list-style-type: none"> ♦ Severe vertigo of sudden onset ♦ Absence of cochlear signs ♦ Spontaneous nystagmus of horizontal type

Investigations

- Hearing test
 - Within normal limits
- Caloric test and electronystagmography
 - Unilateral or bilateral weakness with or without directional preponderance.

Treatment

Supportive	Specific	Vasodilators
	<i>Vestibular sedatives (to relieve vertigo)</i>	
<ul style="list-style-type: none"> ♦ Reassurance and psychological support to allay worry and anxiety ♦ Bed rest with head supported on pillow to avoid excessive movements 	<ul style="list-style-type: none"> ♦ 15–75 mg prochlorperazine daily orally or IM ♦ 25 mg chlorpromazine TID ♦ 5–10 mg diazepam IV (tranquillizing effect and suppresses activity of medial vestibular nucleus) ♦ 0.4 mg atropine SC 	<ol style="list-style-type: none"> Carbogen inhalation (5% CO₂ and 95% O₂) <ul style="list-style-type: none"> – Good cerebral vasodilator and improves labyrinthine circulation Histamine drip <ul style="list-style-type: none"> – 2.75 mg histamine diphosphate dissolved in 500 mL glucose by IV drip

7. Theories of formation of cholesteatoma.

Refer Question No. 1 June 2013 (RS2).

8. Complications of tonsillectomy.

- Tonsillectomy is surgical removal of tonsils.

Indications			
Absolute	Relative	As component of another operation	Contraindications "ABCDEFGH"
<ul style="list-style-type: none"> ♦ Recurrent infections of throat (MC) <ul style="list-style-type: none"> – ≥7 episodes/1 year – 5 episodes/yr for 2 years – 3 episodes/yr for 3 years – ≥2 weeks absence from school or work in a year ♦ Peritonsillar abscess <ul style="list-style-type: none"> – In children 4–6 weeks after treatment – In adults for 2nd attack ♦ Tonsillitis causing febrile seizures ♦ Chronic tonsillitis with obstruction to airway and food passage and difficulty in speech ♦ Suspicious malignancy 	<ul style="list-style-type: none"> ♦ Diphtheria carriers not responding to antibiotics ♦ Streptococcal carriers ♦ Chronic tonsillitis with bad taste or halitosis not responding to antibiotics ♦ Recurrent streptococcal tonsillitis in patients with valvular heart disease ♦ Foreign body embedded in tonsil ♦ Tonsillith ♦ Chronic enlargement of regional lymph nodes ♦ Failure to thrive 	<ul style="list-style-type: none"> ♦ Palatopharyngoplasty (for sleep apnoea) ♦ Glossopharyngeal neurectomy ♦ Removal of styloid process 	<ul style="list-style-type: none"> ♦ Acute infection (upper respiratory infection, tonsillitis) ♦ Bleeding disorders like hemophilia, purpura, leukemia, aplastic anemia ♦ Children under 3 years ♦ Developmental defects (overt of submucous cleft palate) ♦ Epidemic of polio ♦ Female in menses and pregnancy ♦ General systemic disorders like diabetes, hypertension, tuberculosis, asthma ♦ Hemogram <10% (anemia)

Anesthesia

- Usually general anesthesia with endotracheal intubation
- Local anesthesia in adults with 2% lignocaine with 1:100,000 adrenaline infiltrated in peritonsillar tissue or spraying throat with 4% lignocaine.

Position

- Rose's position, i.e. supine position with extended head by placing a pillow under shoulder and head stabilized by a rubber ring
- Surgeon standing on right side of table or sitting at head end of table.

Techniques

Cold methods	Hot method
<ul style="list-style-type: none"> ♦ Dissection and snare (most common) ♦ Guillotine method ♦ Intracapsular tonsillectomy with debrider ♦ Harmonic scalpel ♦ Plasma-mediated ablation technique ♦ Cryosurgical technique 	<ul style="list-style-type: none"> ♦ Electrocautery ♦ Laser tonsillectomy ♦ Coblation tonsillectomy ♦ Radiofrequency

Procedure (Dissection and Snare Method)

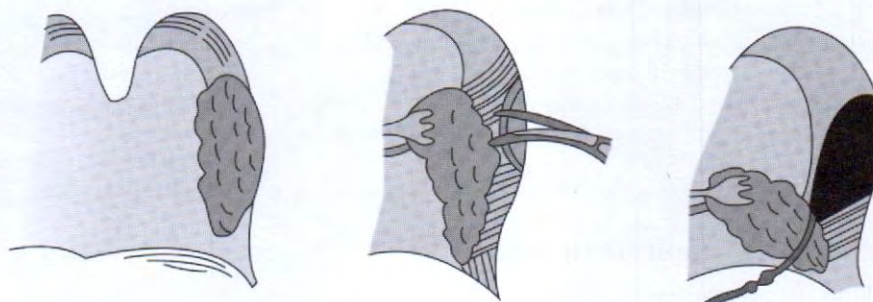


Figure 19: Tonsillectomy

- Keep mouth open using Boyle-Davis mouth gag held in place by Draffin's bipods
- Identify tonsils and grasp them using tonsil holding forceps or Luc forceps
- Exert medial pull on tonsil and make a J-shaped incision on mucosa where it reflects from tonsil to anterior pillar
- Extend incision to posterior border along upper pole
- Dissect tonsil from peritonsillar tissue using blunt curved scissors, separating its upper pole
- Hold tonsils by its upper pole and exert medial and downward traction
- Continue blunt dissection using tonsillar dissector or scissors till lower pole reached
- Loop tonsils with tonsillar snare at its pedicle and crush and cut it to remove tonsil
- Apply pressure in peritonsillar fossa using gauze sponge
- Identify and ligate bleeding points or electrocautery them
- Repeat procedure on other side.

Postoperative Care

i. Immediate general care	<ul style="list-style-type: none"> ◆ Keep patient in lateral position till waning of anesthetic effects to avoid any aspiration ◆ Watch for bleeding from mouth and nose ◆ Check for vitals (pulse, BP, respiration)
ii. Diet	<ul style="list-style-type: none"> ◆ Encourage plenty of fluids ◆ Initially soft nonspicy diet then gradually shifted to solid diet ◆ Cold items like cold milk, ice creams offer relief from pain
iii. Oral hygiene	<ul style="list-style-type: none"> ◆ Gargles with antiseptic solution 3–4 times a day
iv. Analgesics for 1 week	<ul style="list-style-type: none"> ◆ To relieve local pain in throat and referred pain to ear
v. Antibiotics for 1 week	<ul style="list-style-type: none"> ◆ To prevent secondary infection

Complications

<i>Immediate</i>	<i>Delayed</i>
<p>i. Primary hemorrhage</p> <ul style="list-style-type: none"> – Occurs during time of operation <p><i>Cause</i></p> <ul style="list-style-type: none"> – Poor patient selection, improper technique <p><i>Treatment</i></p> <ul style="list-style-type: none"> – Controlled by pressure, ligation or electrocautery of bleeding vessels – Sometimes approximation of pillars with mattress sutures or external carotid artery ligation may be needed <p>ii. Reactionary hemorrhage</p> <ul style="list-style-type: none"> – Occurs within 24 hours <p><i>Cause</i></p> <ul style="list-style-type: none"> – Due to waning of vasoconstrictor effects of adrenaline, slippage of ligature, postoperative rise in BP <p><i>Treatment</i></p> <ul style="list-style-type: none"> – Controlled by removal of clots, application of pressure or vasoconstriction – H₂O₂ gargles helps to remove clots and acts as mild cauterizing agent – In persistent bleeding, ligation or electrocautery of bleeders under general anesthesia <p>iii. Injury to surrounding structures</p> <ul style="list-style-type: none"> – Injury to tonsillar pillars, uvula, soft palate, tongue, teeth or superior constrictor muscle due to bad surgical technique <p>iv. Aspiration of blood</p> <ul style="list-style-type: none"> – Due to wrong positioning <p>v. Facial edema</p> <ul style="list-style-type: none"> – Particularly of eyelids <p>vi. Surgical emphysema (rare)</p> <ul style="list-style-type: none"> – Due to injury to superior constrictor muscle 	<p>i. Secondary hemorrhage</p> <ul style="list-style-type: none"> – Occurs between 5–10th postoperative day <p><i>Cause</i></p> <ul style="list-style-type: none"> – Due to sloughing of arteries by infection and premature separation of membrane <p><i>Treatment</i></p> <ul style="list-style-type: none"> – Controlled by removal of clots, topical application of dilute adrenaline or H₂O₂ with pressure – For profuse bleeding, ligation or electrocautery of bleeders under general anesthesia – Sometimes approximation of pillars with mattress sutures or external carotid artery ligation may be needed – Systemic antibiotics to control infection – Coagulants to enhance coagulation – Severe cases also require blood transfusion <p>ii. Infection</p> <ul style="list-style-type: none"> – Infection of tonsillar fossa leading to parapharyngeal abscess or otitis media <p>iii. Respiratory complications</p> <ul style="list-style-type: none"> – Atelectasis or lung abscess due to aspiration of blood, mucus or tissue fragments <p>iv. Scarring of soft palate and pillars</p> <p>v. Change of voice</p> <ul style="list-style-type: none"> – Nasal twang due to edema and inflammation of palatal muscles (temporary) or excessive damage to soft palate or its fibrosis (permanent) <p>vi. Tonsillar remnants</p> <ul style="list-style-type: none"> – Due inadequate surgery, tags or tonsillar tissue may be left behind which may get secondarily infected <p>vii. Hypertrophy of lingual tonsil</p> <ul style="list-style-type: none"> – Late complication to compensate loss of palatine tonsils

9. Causes of dysphagia.

- Dysphagia is difficulty in swallowing.

Etiology

Preesophageal (disturbance in oral or pharyngeal phase of deglutition)	Esophageal causes (disturbance in esophageal phase of deglutition)	Functional
A. Oral phase a. Disturbance in mastication <ul style="list-style-type: none"> - Trismus - Fracture of mandible - Tumors of upper or lower jaw - Disorders of TM joint b. Disturbance in lubrication <ul style="list-style-type: none"> - Xerostomia following radiotherapy - Milkulicz disease c. Disturbance in mobility of tongue <ul style="list-style-type: none"> - Paralysis of tongue - Painful ulcers - Tumors of tongue - Lingual abscess - Total glossectomy d. Defects of palate <ul style="list-style-type: none"> - Cleft palate - Oronasal fistula e. Lesions of buccal cavity and floor of mouth <ul style="list-style-type: none"> - Stomatitis - Ulcerative lesions - Ludwig's angina B. Pharyngeal phase a. Obstructive pharyngeal lesions <ul style="list-style-type: none"> - Tumors of tonsils, soft palate, base of tongue, supraglottic larynx - Obstructive hypertrophic tonsils b. Inflammatory conditions <ul style="list-style-type: none"> - Acute tonsillitis - Peritonsillar abscess - Retro- or parapharyngeal abscess - Acute epiglottitis - Laryngeal edema c. Spasmodic condition <ul style="list-style-type: none"> - Tetanus - Rabies d. Paralytic condition <ul style="list-style-type: none"> - Paralysis of soft palate due to diphtheria, bulbar palsy, cerebrovascular accidents 	A. In lumen <ul style="list-style-type: none"> - Foreign body - Large bolus B. In wall a. Congenital <ul style="list-style-type: none"> - Tracheoesophageal fistula - Stricture b. Traumatic <ul style="list-style-type: none"> - Corrosive poisoning c. Inflammation <ul style="list-style-type: none"> - Hiatus hernia with lower esophagitis - After exanthemata d. Neoplasms <ul style="list-style-type: none"> - Leiomyoma - Carcinoma of esophagus e. Neurological <ul style="list-style-type: none"> - Paralytic - Spasm of cricopharynx and esophagus - Tetanus - Myasthenia gravis f. Miscellaneous <ul style="list-style-type: none"> - Plummer Vinson syndrome - Achalasia - Hypopharyngeal diverticulum C. Pressure on esophagus a. Cervical <ul style="list-style-type: none"> - Malignancy of thyroid gland - Malignancy of lymph node - Pharyngeal pouch - Cervical spondylosis (cervical osteophytes) b. Thoracic <ul style="list-style-type: none"> - Aneurysm of aorta - Mediastinal tumors - Lymphadenopathy in Hodgkin's disease - Tuberculosis - Cardiac enlargements - Pericardial effusion - Retrosternal goiter - Vascular rings by aberrant blood vessel (dysphagia lusoria) c. Abdominal <ul style="list-style-type: none"> - Hepatic enlargement 	♦ Globus hystericus

Clinical Features

- Any age may be affected depending upon underlying pathology:
 - Achalasia cardia—common in 3rd and 4th decade of life
 - Malignancy seen mostly in elderly above 40 years of age.
- Sexes may also be affected based on etiology:
 - Malignancy common in males
 - Plummer Vinson syndrome exclusively in females.

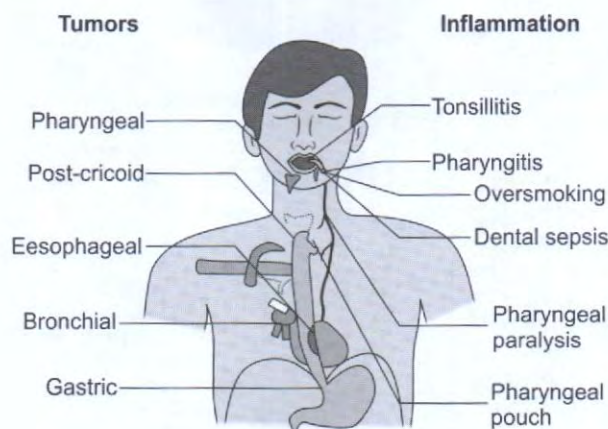


Figure 20: Dysphagia—causes

- History of previous conditions suggesting underlying condition present:
 - History of foreign body ingestion
 - History of corrosive poisoning
 - History neurological conditions like polio, diphtheria, etc.

Symptoms	Signs
<ul style="list-style-type: none"> No symptoms at early cases Acute dysphagia may be due to foreign bodies or acute esophagitis Chronic dysphagia may present as acute dysphagia Dysphagia to solids is presenting feature of strictures and malignancy Dysphagia to liquids is characteristics of achalasia cardia 	<ul style="list-style-type: none"> Vary depending upon underlying etiology

Investigations

a. Radiography

i. X-ray chest	♦ To exclude cardiovascular, pulmonary and mediastinal diseases
ii. Lateral view of neck	♦ To exclude cervical osteophytes and soft tissue involvement of post-cricoid or retropharyngeal space
iii. Barium swallow	♦ Ideal to diagnose malignancy, achalasia, strictures, diverticula, hiatus hernia or esophageal spasms
iv. CT scan	♦ Detects space occupying lesions

b. Manometric and pH studies

- To detect motility disorders, gastroesophageal reflux diseases

c. Endoscopy

- Esophagoscopy, laryngoscopy and bronchoscopy with biopsy

d. Others

- Thyroid scan (for thyroid malignancy)
- Cardiac catheterization (for vascular anomalies).

10. Topodiagnostic tests for facial nerve palsy.

- Topodiagnostic tests are tests to detect site of lesion in lower motor neuron palsy of facial nerve.

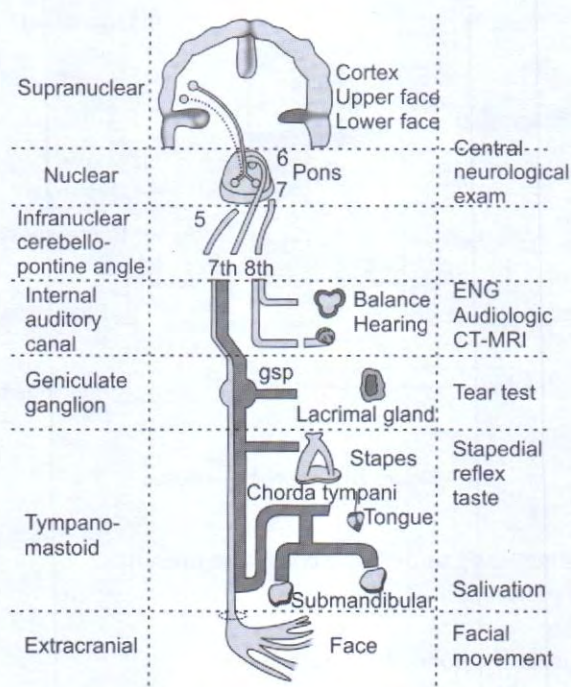


Figure 21: Topodiagnostic tests for facial nerve palsy

	Procedure	Inference
a. Schirmer's test or lacrimation test	<ul style="list-style-type: none"> ♦ Lacrimation on both sides is compared by placing strip of paper in lower fornix of each eye ♦ Measure amount of wetting of strip 	♦ Decreased lacrimation indicates lesion proximal to geniculate ganglion
b. Stapedial reflex	♦ Tested by tympanometry	♦ Loss of stapedial reflex indicates level of lesion above nerve to stapedius
c. Taste test	<ul style="list-style-type: none"> ♦ Place a drop of salt or sugar solution on one side of tongue ♦ Can also be tested by electrogustometry 	♦ Impairment of taste indicates lesion above chorda tympani nerve
d. Submandibular salivary flow test	<ul style="list-style-type: none"> ♦ Pass polythene tube into both Wharton's ducts ♦ Count drops of saliva for period of a minute 	♦ Decreased salivation indicates level of injury above chorda tympani nerve

11. Malignant otitis externa.

- Malignant otitis externa is progressive, necrotizing infection of external ear.

Etiology

Causative agent	Predisposing factors
♦ <i>Pseudomonas aeruginosa</i>	<ul style="list-style-type: none"> ♦ Uncontrolled diabetes (>55 years) ♦ Immunosuppression ♦ Malnutrition ♦ Anemia (in children) ♦ Organ transplant recipient

Clinical Features

- Commonly seen in elderly
- Insidious onset.

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Excruciating pain in external and deep meatus ♦ Mucopurulent discharge initially becoming purulent and bloody later ♦ Hearing loss ♦ Facial nerve palsy 	<ul style="list-style-type: none"> ♦ Sensorineural hearing loss ♦ Doughy feeling pinna ♦ Tenderness on pulling pinna ♦ Narrow external auditory canal with discharge ♦ Rapidly spreading granulations in deep meatus at junction of cartilaginous and bony parts ♦ Sagging of posterior meatal wall ♦ Extensive destruction of tissues in meatus, preauricular and postauricular tissue ♦ Bulging, congestion and granulation of tympanic membrane (involvement of middle ear) ♦ Mastoid tenderness (late cases) ♦ Multiple cranial nerve paralysis (late cases)

Investigations

Ear swab	Blood sugar	CT scan or MRI with gallium contrast
♦ For culture and sensitivity	♦ To confirm diabetes	♦ To know extent of disease

Treatment

Conservative	Operative
a. Antibiotics in high doses both topical and IV for 6–8 weeks <ul style="list-style-type: none"> – Gentamicin/Tobramycin – Ticarcillin – Third generation cephalosporin b. Control of diabetes <ul style="list-style-type: none"> – Insulin or oral antidiabetics 	a. Drainage of subperiosteal abscess b. Surgical debridement of devitalized tissue and bone

Complications

- Multiple cranial nerve paralysis due to spread of infection to skull base and jugular foramen
- Spread of infection to temporomandibular fossa (anteriorly), mastoid (posteriorly), middle ear and petrous bone (medially).

12. Mastoid abscess.

Refer Question No. 4 December 2008 (RS2).

■ SHORT ANSWERS

13. Ludwig's angina.

Refer Question No. 5 December 2011 (RS2).

14. Differences between ethmoid polyp and antrochoanal polyp.

Refer Question No. 3 June 2016 (RS2).

15. Indications for tracheostomy.

Refer Question No. 9 December 2007 (RS2).

16. ENT manifestations in acquired immunodeficiency syndrome.

Refer Question No. 8 December 2007 (RS2).

17. Mucociliary dyskinesia.

- Mucociliary mechanism in nasal cavity is an important defence mechanism to remove any inhaled pathogen.

Mucociliary Dyskinesia

- Mucociliary dyskinesia is a inherited disorder characterized by defective mucociliary mechanism.

Etiology

- Recessively inherited disorder.

Pathogenesis

- Abnormal ciliary activity due to abnormal ciliary ultrastructure.

Clinical Features

- Recurrent lower respiratory tract infections
- Chronic rhinitis, sinusitis and otitis media.

Treatment

- Chest physiotherapy
 - To improve postural drainage and coughing
- Treatment of recurrent infections
 - Prompt treatment of ENT and respiratory infections to prevent further complications.

18. Rinne test.

Refer Question No. 18 December 2007 (RS2).

19. Differences between adult and infant larynx.

Refer Question No. 5 June 2009 (RS2).

20. Applications of LASER in ENT.

- LASER is an acronym for Light Amplification by Stimulated Emission of Radiation.

Types of LASERs (Used in ENT)

	Properties	Advantages
a. CO ₂ lasers (Most commonly used)	<ul style="list-style-type: none"> ♦ Wavelength of 10600 nm ♦ Requires aiming beam of helium-neon laser (cannot pass through flexible fibroscope) ♦ Penetration depth of 0.1 mm 	<ul style="list-style-type: none"> ♦ Very effective to vaporize tissues ♦ Immediate tissue destruction ♦ Precise and clean surgery ♦ Minimal damage to surrounding tissue ♦ Bloodless field by sealing small blood vessels ♦ Rapid healing ♦ Can be used in patients with bleeding dyscrasias and coagulopathies
b. Argon laser	<ul style="list-style-type: none"> ♦ Wavelength of 488–514 nm ♦ Blue in color ♦ Pass through clear fluid but gets absorbed by pigmented tissues 	<ul style="list-style-type: none"> ♦ Fall in visible spectrum, hence do not require separate beam to focus them, therefore, ideal for microsurgery of ear like stapedotomy, lysis of middle ear adhesions, spot welding of tympanoplasty grafts, etc.
c. KETP-532 laser (Potassium titanyl phosphate)	<ul style="list-style-type: none"> ♦ Wavelength of 532 nm ♦ Blue-green in color ♦ Selectively absorbed by pigments ♦ Used for microsurgery of ear 	

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	Properties	Advantages
d. Nd:YAG laser (Neodymium-yttrium aluminium garnet)	<ul style="list-style-type: none"> ♦ Wavelength of 1060 nm ♦ Transmitted by flexible endoscopes ♦ Penetration depth of 4 mm ♦ Used for endobronchial surgery like coagulation of tracheal and endobronchial tumors 	
e. Ho:YAG laser	<ul style="list-style-type: none"> ♦ Wavelength of 2100 nm ♦ Delivered by optical fibers ♦ Short penetration depth ♦ Used selectively for otolaryngology, i.e. facial nerve decompression, endoscopic sinus surgery 	

Applications (Indications)

- a. Ear
 - Stapedectomy, ossicular sculpturing, lysis of adhesions and granulation tissue, control of bleeding.
- b. Nose
 - Papillomas, rhinophyma, telangiectasias, nasal polypi, choanal atresia, turbinectomy, inferior turbinoplasty, epistaxis.
- c. Oral cavity
 - Multiple areas of leukoplakia, erythroplakia, small superficial cancers, debulking of large, recurrent or inoperable tumors.
- d. Oropharynx
 - Tonsillar and pharyngeal tumors
 - Laser tonsillectomy possible in patients with coagulopathy or hypertension.
- e. Larynx
 - Papilloma larynx, laryngeal web, subglottic stenosis, capillary hemoangioma
 - Microlaryngeal surgery for vocal nodule, leukoplakia of cord, papilloma, polypoid degeneration of cord, endoscopic laser arytenoidectomy, malignant T₁ lesions of vocal cord.
- f. Trachea and bronchi
 - Recurrent papillomatosis, tracheal stenosis, granulation tissues and bronchial adenoma, debulking of obstructive malignant lesions of trachea or bronchii.
- g. Plastic surgery
 - Benign and malignant tumors of skin, vaporization of naevi and tattoos.
- h. Neuro-otology
 - Removal of acoustic neuroma.

Complications	Advantages	Disadvantages
<ul style="list-style-type: none"> ♦ Damage to eyes due to exposure (can be prevented by use of protective eye goggles) ♦ Damage to anesthetic tubes resulting in explosion (can be prevented by protecting them with metal foils or use of flexible metallic tubes) ♦ Burning of exposed skin ♦ Damage to vocal cords ♦ Tracheal perforation 	<ul style="list-style-type: none"> ♦ Precision surgery ♦ Minimal bleeding ♦ Minimal tissue reaction ♦ Less postoperative edema and pain 	<ul style="list-style-type: none"> ♦ Costly ♦ Biopsy is not possible ♦ Trauma to patient and doctor

21. Rhinitis medicamentosa.

Refer Question No. 19 June 2009 (RS2).

22. Diagnostic nasal endoscopy.

Refer Question No. 22 June 2009 (RS2).

MBBS PHASE III EXAMINATION

JUNE 2011

(Revised Scheme 2)

■ LONG ESSAYS

1. Discuss the etiology, clinical features and management of acute coalescent mastoiditis.

Refer Question No. 3 December 2009 (RS2).

2. Discuss the etiology, clinical features and management of juvenile nasopharyngeal angiofibroma.

Refer Question No. 1 December 2009 (RS2).

■ SHORT ESSAYS

3. Le Fort fractures.

Refer Question No. 3 December 2010 (RS2).

4. Management of Meniere's disease.

Refer Question No. 2 December 2008 (RS2).

5. Otogenic causes of facial palsy.

Refer Question No. 11 December 2008 (RS2).

6. Indications and complications of tracheostomy.

Refer Question No. 9 December 2007 (RS2).

7. Clinical features and management of peritonsillar abscess.

- Collection of pus in peritonsillar space (outside tonsillar capsule and medial to superior constrictor muscle) is called peritonsillar abscess or quinsy.

Types

<i>Anterior presentation (MC)</i>	<i>Posterior presentation</i>	<i>Lingual</i>	<i>Tonsillar</i>
Abscess points through anterior pillar	Abscess pushes tonsil forwards	Affects lower pole of tonsil	Abscess within tonsil

Etiopathogenesis

Causative agents	Predisposing factors	Pathogenesis
<ul style="list-style-type: none"> ♦ <i>Streptococcus pyogenes</i> ♦ <i>Staphylococcus aureus</i> ♦ Pneumococci ♦ Anaerobic organisms 	<ul style="list-style-type: none"> ♦ Recurrent tonsillitis (usually as a sequel) ♦ Foreign body embedded in tonsillar substance ♦ Tonsilloliths or cyst ♦ Dental infection like periodontitis ♦ Inflammation of Weber gland (accessory salivary gland just above superior pole in soft palate) 	<p>Recurrent tonsillitis</p> <p>↓</p> <p>Fibrosis of mouths of tonsillar crypts (esp. cista magna)</p> <p>↓</p> <p>Bacterial infection of tonsils</p> <p>↓</p> <p>Edema and fibrotic stenosis of mouth of crypts</p> <p>↓</p> <p>Sealing of tonsillar crypts</p> <p>↓</p> <p>Formation of intratonsillar abscess</p> <p>↓</p> <p>Bursting of abscess through tonsillar capsule</p> <p>↓</p> <p>Peritonsillitis</p> <p>↓</p> <p>Peritonsillar abscess</p>

Pathology

- Fibrosis of intratonsillar crypts.

Clinical Features

- Usually seen in adult males aged 20–50 years
- Mostly unilateral but occasionally bilateral.

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Unilateral pain in throat, aggravated on swallowing ♦ Painful swallowing (odynophagia) → saliva dribbling from angle of mouth ♦ Muffled and thick speech (hot potato voice) ♦ Foul breath (due to sepsis in oral cavity) ♦ Earache on same side (referred pain via 9th cranial nerve) ♦ Difficulty in opening mouth (due to spasm of pterygoid muscles) ♦ Constitutional symptoms like fever, chills and rigor, general malaise, body aches, headache, nausea and constipation 	<ul style="list-style-type: none"> ♦ Congestion and swelling of tonsils, pillars and soft palate on involved side ♦ Swollen, elongated and edematous uvula pushed to opposite side ♦ Bulging of soft palate and anterior pillar above tonsil ♦ Tonsils pushed medially and downwards ♦ Tonsillar region covered with mucopus ♦ Purulent fetid discharge if abscess is ruptured ♦ Enlarged jugulodigastric lymph nodes ♦ Trismus ♦ Torticollis towards side of abscess

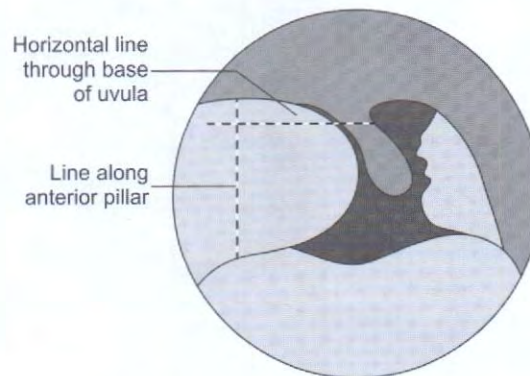


Figure 1: Peritonsillar abscess—signs

Investigations

Blood investigations	Blood sugar	Culture and sensitivity	CT scan
♦ Indicates septicemia	♦ To rule out diabetes mellitus	♦ To identify causative organism and its antibiotic sensitivity	♦ To determine extent of abscess and presence of parapharyngeal abscess if any

Differential Diagnosis

Acute tonsillitis	Retropharyngeal or parapharyngeal abscess	Malignancy of tonsils (Lymphoepithelioma)
♦ Bilateral ♦ Less severe pain ♦ No radiating pain to ear		♦ More chronic ♦ Less pain ♦ Hard to feel ♦ No pus on incision

Treatment

Supportive	Conservative	Operative
♦ Hospitalization and bed rest ♦ Parenteral nutrition ♦ Oral hygiene by regular mouth wash by H ₂ O ₂ or saline ♦ Analgesics to relieve pain and reduce fever	a. Antibiotics – IV infusion of broad spectrum antibiotics	a. Incision and drainage (described below) b. Tonsillectomy <i>Procedures</i> i. Abscess or hot tonsillectomy – Removal of tonsils including abscess <i>Disadvantages</i> – Risk of rupture of abscess during anaesthesia – Excessive bleeding ii. Interval tonsillectomy – Removal of tonsils after 4–6 weeks following remission

- Incision and drainage (most preferred)

Indications

- Formation of frank abscess

Anesthesia

- Local anesthesia with 4% lignocaine
- General anesthesia with precautions in uncooperative children

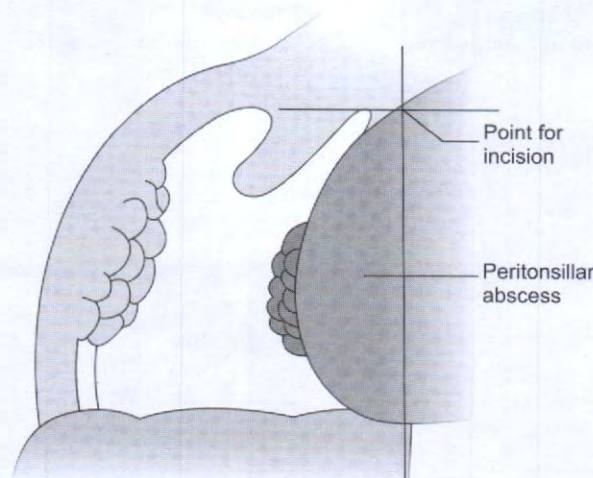
Incision (any of the following)

Figure 2: Peritonsillar abscess—point of incision

- On point of maximum bulge above upper pole of tonsil
- At intersection of imaginary horizontal line drawn through base of uvula and vertical line along base of anterior pillar
- Through intratonsillar cleft.

Procedure

- Make a small stab incision with quinsy forceps or a guarded knife (guarded by winding adhesive tape at distance of 1 cm from its tape)
- Insert a sinus forceps during the procedure to open and drain abscess and may be done on following day to drain any reaccumulations.

Follow-up

- H₂O₂ gargles
- Antibiotics and analgesics.

Complications (of quinsy)

- Parapharyngeal abscess
- Supraglottic edema
- Septicemia
- Pneumonitis or lung abscess
- Jugular vein thrombosis or phlebitis
- Spontaneous hemorrhage from carotids or jugular due to necrosis.

8. Differential diagnosis of lateral neck swellings.

- Swellings in neck could be due to various reasons.

Differential Diagnosis of Lateral Neck Swellings

<i>In submandibular triangle</i>	<i>In carotid triangle</i>	<i>In posterior triangle</i>
a. Enlarged submandibular lymph node <ul style="list-style-type: none"> – Common – Painful, tender and soft enlargement—acute lymphadenitis – Firm and matted—tuberculosis – Hard, with or without fixity—secondaries – Firm and rubbery—Non Hodgkin's lymphoma 	a. Branchial cyst <ul style="list-style-type: none"> – Common – Smooth surface and round borders – Soft, cystic, fluctuant and transillumination negative – Sternomastoid contraction test is positive 	a. Enlarged supraclavicular lymph node <ul style="list-style-type: none"> – Painful, tender and soft enlargement—acute lymphadenitis – Firm and matted—tuberculosis – Hard, with or without fixity—secondaries – Firm and rubbery—non-Hodgkin's lymphoma
b. Enlarged submandibular salivary gland <ul style="list-style-type: none"> – Common – Irregular or nodular swelling – Bidigitally palpable mass 	b. Lymph node swelling (cold abscess) <ul style="list-style-type: none"> – In young patients – Multiple lymph nodes with or without fever 	b. Cystic hygroma <ul style="list-style-type: none"> – Present since birth – Smooth, soft, fluctuant, compressible, brilliantly transilluminant swelling
c. Deep or plunging ranula (not uncommon) <ul style="list-style-type: none"> – Soft, cystic, fluctuant swelling, brilliantly transilluminant swelling – Bluish in color – Cross fluctuation test positive 	c. Aneurysm of carotid artery <ul style="list-style-type: none"> – Tensely cystic, fluctuant, transillumination negative swelling with expansile pulsation – Compressibility is positive – Bruit and thrill is present 	c. Pharyngeal pouch <ul style="list-style-type: none"> – Located behind sternomastoid below level of thyroid cartilage – Soft swelling which can be emptied
d. Ludwig's angina (not uncommon) <ul style="list-style-type: none"> – Brawny edema/diffuse swelling – Intraoral edema in floor of mouth – Putrid halitosis – Severe toxicity 	d. Thyroid swelling	d. Subclavian aneurysm <ul style="list-style-type: none"> – Pulsatile swelling
e. Lateral sublingual dermoid cyst (rare) <ul style="list-style-type: none"> – Seen during childhood and later in life – Painless, slow growing swelling 	e. Carotid body tumor (rare) <ul style="list-style-type: none"> – Smooth surface or lobulated with round borders – Oval, vertically placed swelling – Firm to hard consistency 	e. Cervical rib <ul style="list-style-type: none"> – Asymptomatic – Hard, fixed bony mass – Pain aggravated during work, exercise and relieved by rest – Wasting of hand and forearm muscles
	f. Laryngocele (very rare) <ul style="list-style-type: none"> – Smooth, oval, boggy swelling moving upward on swallowing 	f. Lipoma <ul style="list-style-type: none"> – Localized, lobular, nontender, semifluctuant swelling

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<i>In submandibular triangle</i>	<i>In carotid triangle</i>	<i>In posterior triangle</i>
<ul style="list-style-type: none"> – Soft, cystic, fluctuant, transillumination negative swelling – Underlying bony defect <p>f. Extension of tumor growth from jaw (rare)</p>	<ul style="list-style-type: none"> – Expansile cough impulse present – Tympanitic note on percussion <p>g. Sternomastoid tumor (rare)</p> <ul style="list-style-type: none"> – Seen in infants or children – Firm to hard, 1–2 cm swelling in midline of sternomastoid – Tender and mobile sideways – Associated with torticollis <p>h. Neurofibroma of vagus</p> <ul style="list-style-type: none"> – Vertically placed oval swelling – Firm to hard consistency – Dry cough and bradycardia on pressure over swelling 	<ul style="list-style-type: none"> – Mobile with edges of slipping between palpating fingers (slip sign) <p>g. Cold abscess</p> <ul style="list-style-type: none"> – No evidence of signs of inflammation – Not warm, nontender, smooth, soft and fluctuant, non transilluminating swelling – Not adherent to skin, no redness – Contains cheesy material

9. Foreign body in bronchus.

- Small foreign bodies ingested accidentally may lodge anywhere in bronchus after passing through vocal cords.

Etiology

<i>Etiology</i>	<i>Predisposing factors</i>
<ul style="list-style-type: none"> ♦ Accidental ♦ Intentional in psychiatric patients 	<p>a. Interference with deglutition reflex</p> <ul style="list-style-type: none"> – Due to coughing, crying or tapping on back while swallowing may lead to inhalation of food into bronchi <p>b. Altered sensorium</p> <ul style="list-style-type: none"> – Inhalation of fluid, food is seen in unconscious, drunken and anesthetic people <p>c. Neurological imbalance</p> <ul style="list-style-type: none"> – Paralysis of pharynx or larynx <p>d. Improper mastication</p> <ul style="list-style-type: none"> – Hurried swallowing of improperly masticated food may lead to inhalation of food

Nature of Foreign Bodies

<i>Nonirritating type</i>	<i>Irritating type</i>
<ul style="list-style-type: none"> ♦ Less irritant ♦ Remain symptomless for long time ♦ Cause granuloma-hemoptysis ♦ For example, plastic, glass or metallic objects 	<ul style="list-style-type: none"> ♦ More toxic ♦ Sets up a diffuse violent reaction leading to congestion and edema of tracheobronchial mucosa (vegetal bronchitis) ♦ Cause airway obstruction by swelling up and later causing suppuration in lungs ♦ For example, groundnut, seeds of fruits like chikoo, custard apple, grams

Clinical Features

- Common in children esp. below 4 years of age.

<i>Stage of inhalation</i>	<i>Latent stage</i>	<i>Stage of manifestations</i>
<ul style="list-style-type: none"> ♦ Lasts for a short time ♦ Characterized by choking, gagging and wheezing ♦ Patient become cynosed ♦ Death may occur in case of large foreign bodies ♦ Foreign body may be coughed out or lodge in tracheobronchial tree 	<ul style="list-style-type: none"> ♦ Lasts for variable period depending upon size and nature of foreign body (shorter in larger and vegetable foreign bodies) ♦ Disappearance of initial symptoms as respiratory mucosa adopts to foreign body 	<ul style="list-style-type: none"> ♦ Caused by obstruction to airway, inflammation or trauma induced by foreign body ♦ Foreign bodies usually enter right bronchus because it is wider and more in line with tracheal lumen <ol style="list-style-type: none"> Wheeze <ul style="list-style-type: none"> – Caused by partial obstruction Atelectasis <ul style="list-style-type: none"> – Due to total obstruction of lobar or segmental bronchus, thus preventing entry of air but only exit

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Stage of inhalation	Latent stage	Stage of manifestations
		iii. Obstructive emphysema - Due to producing check valve obstruction allowing only entry of air but no exit iv. Acute laryngotracheobronchitis - Due to infection or chemical irritation usually caused by vegetable foreign bodies

Investigations

a. X-ray chest (PA and lateral view)	<ul style="list-style-type: none"> May demonstrate radio-opaque foreign bodies May be normal or show atelectasis, emphysema or mediastinal shift (if taken at end of inspiration and expiration)
b. CT scan	<ul style="list-style-type: none"> May be useful
c. Fluoroscopy	<ul style="list-style-type: none"> During inspiration and expiration
d. Bronchogram	<ul style="list-style-type: none"> To delineate radiolucent foreign bodies
e. Bronchoscopy	<ul style="list-style-type: none"> Dual benefit of diagnostic and therapeutic procedure

Differential Diagnosis

- Acute laryngotracheobronchitis
- Pneumonia
- Bronchopneumonia
- Atelectasis.

Treatment

Conservative	Operative
a. Antibiotics - To prevent secondary infection b. Steroids - Started at earliest in full dose to prevent laryngeal edema following prolonged bronchoscopy	a. Bronchoscopy - Treatment of choice - Confirms diagnosis and aids removal of foreign body - Failure to perform bronchoscopy may be much more disastrous than risk involved in performing bronchoscopy (Jackson's dictum) b. Tracheostomy <i>Indications</i> - Laryngeal stridor - Laryngeal edema - To reduce dead space - For inserting large bronchoscope - Removal of large swollen foreign body (obstructing at vocal cords) - Tracheobronchial toilet for secretions c. Thoracotomy and bronchotomy - Indicated when foreign body cannot be removed by bronchoscopy d. Lobectomy or pneumonectomy - Indicated in old neglected foreign bodies causing severe damage to lobe of lung or entire lung

10. Theories of hearing.

Refer Question No. 1 December 2007 (RS2).

11. Anatomy of the middle ear cleft.

Refer Question No. 1 June 2012 (RS2).

12. Clinical features and management of septal perforation.

- Perforation of nasal septum is usually asymptomatic.

Etiology

Trauma (most common)	Pathological	Addiction	Idiopathic
<ul style="list-style-type: none"> ♦ Injury to mucosal flaps during SMR ♦ Cauterization of septum with chemicals or galvanocautery for epistaxis ♦ Thermal or radiational trauma ♦ Mechanical injury due to road traffic accident, assault, etc. ♦ Habitual nose picking ♦ Cosmetic for wearing ring 	<ul style="list-style-type: none"> ♦ Septal abscess ♦ Nasal myiasis ♦ Neoplasm of septum ♦ Pressure necrosis due to rhinolith or neglected foreign body ♦ Chronic granulomatous conditions like lupus, tuberculosis, leprosy, syphilis ♦ Wegener's granuloma 	<ul style="list-style-type: none"> ♦ Cocaine addict 	



Figure 3: Nasal septum perforation

Clinical Features

- Whistling/hissing sound during respiration
- Crust formation over large perforations causing nasal obstruction and severe epistaxis on removal.

Investigations

- Biopsy
 - From granulation or edges of perforation to identify etiology.

Treatment

Conservative	Operative
<ul style="list-style-type: none"> ♦ Alkaline nasal douches to keep nose crust free ♦ Application of bland ointment 	<ul style="list-style-type: none"> ♦ Implantation of thin silastic button for small perforations ♦ Surgical closure by nasal, buccal or skin flaps for larger perforation

SHORT ANSWERS**13. Rhinophyma.**

- Rhinophyma is a slow growing benign tumor of external nose
- Also called potato nose deformity, elephantiasis of nose, cystadenofibroma of nose.

Etiology	Pathology
<ul style="list-style-type: none"> ♦ Acne rosacea (long standing) 	<ul style="list-style-type: none"> ♦ Fibrosis and hypertrophy of sebaceous glands of tip of nose

Clinical Features

- Mostly affects men over middle age.

<i>Symptoms</i>	<i>Signs</i>
<ul style="list-style-type: none"> ♦ Large unsightly mass over nose ♦ Obstruction in breathing ♦ Difficulty to see due to tumor 	<ul style="list-style-type: none"> ♦ Pink, lobulated mass over nose with superficial vascular dilatation ♦ Overlying skin is coarse and pitted

Treatment*Operative*

- Shaving of excessive sebaceous tissue without traumatizing underlying nasal cartilage using sharp knife or CO₂ laser.

14. Vocal polyp.

Refer Question No. 22 June 2008 (RS2).

15. Ranula.

- Ranula is a mucus retention cyst in the floor of the mouth.

Etiology

- Obstruction of ducts of sublingual salivary glands.

Clinical Features

<i>Symptoms</i>	<i>Signs</i>
<ul style="list-style-type: none"> ♦ Swelling in the floor of the mouth ♦ Difficulty in chewing and swallowing 	<ul style="list-style-type: none"> ♦ Bluish, translucent cyst in the floor of the mouth on one side of tongue ♦ Fluctuant ♦ Pushes tongue up ♦ Resembles belly of a frog (Lt. rana = frog)

Treatment*Operative*

- Excision
 - For small cysts confined to mouth.
- Marsupialization
 - For large cysts and plunging ranula.

Complications*Plunging Ranula*

- Enlarged cyst present in neck after penetration of mylohyoid muscle
- It is intraoral ranula with cervical extension and is cross fluctuant across mylohyoid.

Pathogenesis

- Usually arises from both submandibular and sublingual salivary glands as mucus retention cyst initially, which reaches neck by passing across mylohyoid muscle.

Features

- Presents as soft, fluctuant, nontender, dumbbell shaped swellings in submandibular region
- It is bidigitally palpable.

Investigations

- Ultrasound and MRI are diagnostic.

Treatment

- Surgical excision through neck approach along excision of submandibular and sublingual salivary glands.

16. Branchial cyst.

- Branchial cyst is a congenital anomaly due to defective development of pharyngeal/branchial pouches.

Etiology

- Defective development of 1st or 2nd branchial pouch (latter being more common).

Location

- Common in upper and lateral part of neck deep to upper 1/3rd of anterior border of sternomastoid and beneath angle of mandible.

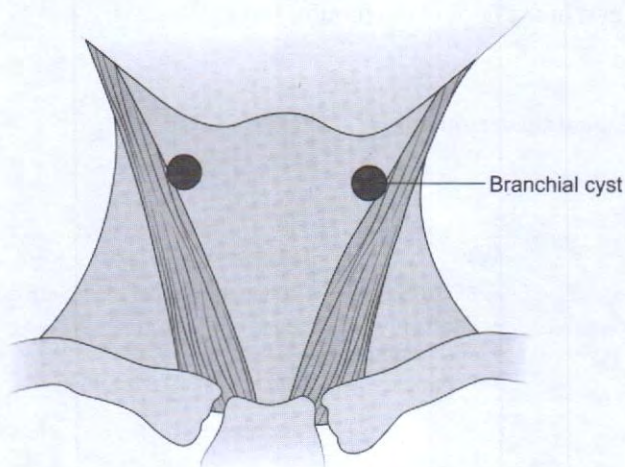


Figure 4: Branchial cyst—location

Clinical Features

- Seen predominantly in young females usually aged between 20–25 years.

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Painless swelling in upper and lateral part of neck ♦ Painful if infected 	<ul style="list-style-type: none"> ♦ Mostly oval but sometimes round shaped swelling ♦ Soft in consistency ♦ Cannot be compressed or reduced ♦ Positive fluctuation test ♦ Negative transillumination

Investigations

- Radiological
 - Contrast X-ray (fistulogram).
- Fine needle aspiration cytology
 - Shows cholesterol crystals.

Treatment*Operative*

- Excision
 - Treatment of choice.

Procedure

- Make incision parallel to skin crease
- Step ladder incision to expose cyst/fistula
- Inject methylene blue in cyst to aid in cyst identification
- Aspirate some amount of fluid to grasp cyst wall
- Excise complete cyst.

Complications

- Infection
- Bronchial fistula.

17. Exostosis.

- Exostosis is a benign tumor of compact bone of external auditory canal.

Etiology

- Repeated entry of cold water entry in meatus (divers, swimmers)
- Chronic suppurative otitis media (discharge may stimulate exostosis formation).

Clinical Features

- More common in males (3 times).

<i>Symptoms</i>	<i>Signs</i>
<ul style="list-style-type: none"> ♦ Blocked sensation in ear ♦ Irritating and itching due to wax collection ♦ Deafness (if external canal completely blocked) 	<ul style="list-style-type: none"> ♦ Multiple, bilateral, smooth, hard, sessile bony swellings in deeper part of meatus near tympanic membrane ♦ Arise from any wall of external canal ♦ Difficulty to visualize tympanic membrane ♦ Certain degree of conductive deafness

Treatment

- Excision
 - Indicated in large, symptomatic exostoses
 - Removed using high speed drill.

18. Rinne's test.

Refer Question No. 18 December 2007 (RS2).

19. Rhinitis sicca.

- Rhinitis sicca is an inflammation of nasal cavity characterized by drying and crusting in anterior 1/3rd of nose.

Etiology

<i>Causative factors</i>	<i>Precipitating factors</i>
<ul style="list-style-type: none"> ♦ Dry, hot and dust environment (promotes drying of nasal secretions) ♦ Certain occupations like farmers, mines, bakers, goldsmiths 	<ul style="list-style-type: none"> ♦ Alcohol ♦ Nutrition ♦ Nasal surgery

Pathology

- Squamous metaplasia of ciliated columnar epithelium
- Atrophy of seromucinous glands
- Periglandular fibrosis
- Viscid and stagnant mucous blanket
- Crust formation.

Clinical Features

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Nasal obstruction ♦ Irritation in nose ♦ Discharge of dirty black crusts ♦ Bleeding from nose 	<ul style="list-style-type: none"> ♦ Drying in anterior 1/3rd of nose ♦ Dirty black crusts over anterior end of inferior turbinate, middle meatus and septum ♦ Epistaxis due to ulceration in nose caused by picking of scabs using fingernails ♦ Septal perforation due to deeper ulcerations caused by picking of scabs

Treatment

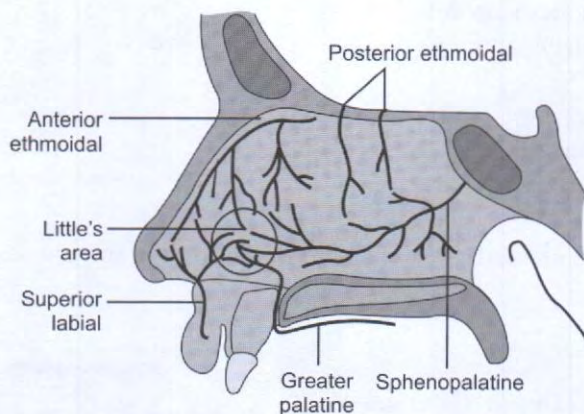
Supportive	Conservative
<ul style="list-style-type: none"> ♦ Change in environment or occupation ♦ Avoiding nasal picking 	<ol style="list-style-type: none"> Lubrication of nose <ul style="list-style-type: none"> – Using vaseline, ghee, oil or butter to diminish scab formation – 25% glucose in glycerine Topical steroids or antibiotics Alkaline nasal douching <ul style="list-style-type: none"> – Douching with warm water containing a teaspoon full of alkaline nasal douch mixture to loosen scabs and remove them – Mixture contains sodium bicarbonate (50 g) + sodium baborate (50 g) and sodium chloride (100 g)

20. Little's area.

- Little's area is a vascular rich area consisting of anastomosis of four arteries supplying nasal septum
- Also called Kiesselbach's plexus.

Location

- In anterior inferior part of nasal septum, just above vestibule.

**Figure 5: Little's area**

Components

- Anterior ethmoidal, a branch of ophthalmic artery
- Septal branch of superior labial artery, a branch of facial artery
- Septal branch of sphenopalatine artery, a branch of maxillary artery
- Greater palatine artery, a branch of maxillary artery.

Applied Anatomy

- Little's area is exposed to drying effect of inspiratory current and to finger nail trauma and, thus, is commonest site for epistaxis in children and young adults
- It is also the site for origin of bleeding polypus (hemangioma) of nasal septum.

21. Waldeyer's ring.

Refer Question No. 3 June 2015 (RS2).

22. Reinke's edema.

Refer Question No. 20 June 2010 (RS2).

MBBS PHASE III EXAMINATION

DECEMBER 2011

(Revised Scheme 2)

LONG ESSAYS

1. Discuss in detail the etiology, pathology, clinical features and management of tubotympanic type of chronic suppurative otitis media.

- Tubotympanic type of chronic suppurative otitis media is long standing chronic infection of anteroinferior part of middle ear cavity, characterized by permanent central perforation and irreversible pathological changes
- Also called safe or benign type because of no risk of serious complications.

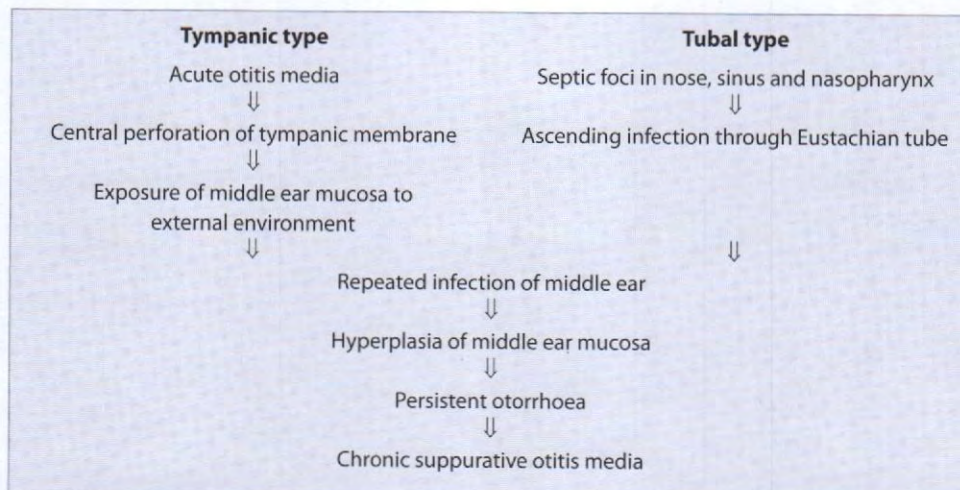
Etiology

<i>Causative organisms</i>	<i>Predisposing factors</i>
<ul style="list-style-type: none">♦ <i>Pseudomonas aeruginosa</i>♦ <i>Proteus</i>♦ <i>E. coli</i>♦ <i>Staphylococcus aureus</i>♦ <i>Bacteroides fragilis</i>♦ Anaerobic streptococci	<ul style="list-style-type: none">♦ Persistence of acute otitis media♦ Acute necrotic otitis media following low resistance due to exanthematous fever (chicken pox, measles, typhoid, etc.)♦ Infection of tonsils, adenoids and infected sinuses (ascending infection)♦ Allergy to ingestants like milk, egg, etc.

Types

<i>Tubal type</i>	<i>Tympanic type (persistent perforation syndrome)</i>
<ul style="list-style-type: none">♦ Infection ascends through Eustachian tube♦ Usually bilateral♦ Common in children	<ul style="list-style-type: none">♦ Infection enters through perforated tympanic membrane♦ Usually unilateral♦ Common in adults

Pathogenesis

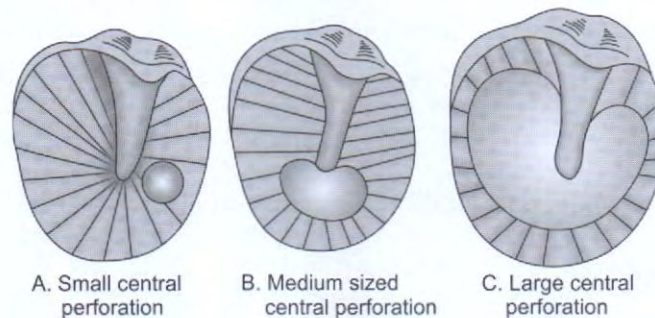


Stages

Active	Quiescent	Inactive	Healed
Active ear discharge	No ear discharge for <3–6 months	No ear discharge >6 months	Healed central perforation

Clinical Features

Symptoms	Signs
<p>a. Ear discharge</p> <ul style="list-style-type: none"> – Nonoffensive, mucoid or mucopurulent, constant or intermittent – White or yellowish – Copious, tenacious – Appears mostly at time of URTI or water entry into ear <p>b. Hearing loss</p> <ul style="list-style-type: none"> – Mild to moderate (rarely exceeds 50 dB) – Conductive hearing loss initially, but later develops sensorineural hearing loss in long standing cases due to cochlear damage by absorbed toxins – Sometimes, patients hear better in presence of ear discharge (round window shielding effect) due to maintenance of phase differential by discharge 	<p>a. Tympanic membrane perforation</p> <ul style="list-style-type: none"> – Central perforation – Anterior, posterior or inferior to handle of malleus – Large (50–75%) > medium (25–50%) > small (<25%) <p>b. Middle ear mucosa</p> <ul style="list-style-type: none"> – Pale pink and moist normally – Red, edematous, velvety and swollen if inflamed <p>c. Aural polyp</p> <ul style="list-style-type: none"> – Smooth pale polyp due to prolapse of edematous and inflamed mucosa – Protrudes through perforation into external canal <p>d. Ossicular chain</p> <ul style="list-style-type: none"> – Usually intact and mobile – Some degree of necrosis, particularly long process of incus <p>e. Tympanosclerosis</p> <ul style="list-style-type: none"> – Chalky white deposits on promontory, ossicles, joints, tendons and round and oval window due to hyalinization and subsequent calcification of subepithelial connective tissue in tympanic membrane and under middle ear mucosa <p>f. Fibrosis and adhesions</p> <ul style="list-style-type: none"> – Due to healing process <p>g. Underlying disease</p> <ul style="list-style-type: none"> – Examination may reveal underlying pathology like infected tonsils, adenoids, sinuses, etc.

**Figure 1:** Tympanic membrane perforation**Investigations**

- Examination under microscope
 - To assess presence of granulations, in growth of squamous epithelium from edges of perforation, status of ossicular chain, tympanosclerosis and adhesions.
- Swab of ear discharge
 - For culture and sensitivity.
- Radiology
 - Mastoid X-ray shows sclerosis but pneumatized mastoid with clouding of air cells
 - X-ray of paranasal sinuses may show sinusitis.

- d. Hearing tests
- Assess type and degree of hearing loss
 - i. Tuning fork tests
 - Rinne's negative
 - Weber lateralized to affected side
 - Absolute conduction test normal
 - ii. Pure tone audiometry
 - Mild-to-moderate hearing loss (<60 dB).

Differential Diagnosis

<i>Acute otitis media</i>	<i>Traumatic perforation</i>	<i>Tuberculous otitis media</i>
<ul style="list-style-type: none"> ♦ Follows upper respiratory tract infection ♦ Passes through different stages ♦ Present small perforation in antero-inferior quadrant 	<ul style="list-style-type: none"> ♦ History of injury ♦ Ragged edges 	<ul style="list-style-type: none"> ♦ Multiple perforations of tympanic membrane ♦ Pale granulations ♦ Painless

Treatment

Supportive

- a. Precautions
- Keep ear dry
 - Avoid hard blowing of nose
 - Use rubber inserts in ear while bathing.
- b. Aural toilet
- Dry mopping of ear with absorbent cotton, suction or irrigation to remove all debris from ear.

Specific

Conservative

- a. Antibiotics
- i. Topical
 - Neomycin, polymyxin, chloromycetin or gentamicin ear drops 3-4 times/day followed by pressure on tragus to force antibiotic into middle ear.
 - ii. Systemic
 - Indicated in acute exacerbations.
- b. Topical steroids
- Used along with antibiotic ear drops for local anti-inflammatory effect.
- c. Antiseptic ear drops
- 1.5% acetic acid to achieve acidic pH in middle ear.
- d. Chemical cautery
- Indicated in small perforations
 - 50% trichloroacetic acid applied to epithelialized edges followed by application of moistened cigarette paper once a week till perforation heals (maximum three sittings).
- e. Treatment of underlying cause
- Diagnose and treat septic foci in tonsils, adenoids or sinuses.

Operative

- a. Aural polypectomy
- Aural polyp or granulations removed using microscope under general anesthesia
 - Done before conservative treatment to facilitate it.
- b. Tympanoplasty
- Tympanoplasty is an operative procedure to eradicate middle ear disease pathology and reconstruct hearing mechanism.

Grafts used

For tympanic membrane (myringoplasty)	For ossicles (ossiculoplasty)
i. Autografts <ul style="list-style-type: none"> – Temporalis fascia (most common) – Perichondrium from tragus or nasal concha – Vein graft – Skin (not used nowadays) ii. Homografts <ul style="list-style-type: none"> – Lyophilized dura – Perichondrium – Tympanic membrane (from cadaver) 	i. Autografts <ul style="list-style-type: none"> – Ossicles (incus, malleus) – Cartilage (tragus, concha) – Bone (spine of Henle, mastoid bone) ii. Homograft <ul style="list-style-type: none"> – Ossicles – Cartilage – Bone iii. Biomaterials <ul style="list-style-type: none"> – Plastics – Ceramics – Teflon – Gold

Indications	Contraindications
<ul style="list-style-type: none"> ♦ Conductive deafness with hearing loss >40 dB ♦ Benign and dry perforation of tympanic membrane 	<ul style="list-style-type: none"> ♦ Residual disease like cholesteatoma ♦ Ear diseases with intracranial complications ♦ Malignant ear diseases ♦ High-risk patients like hypertension, diabetes

Types (Wullstein's classification)

Type	Pathology	Repair
i. Type I (myringoplasty)	Perforated tympanic membrane with intact and mobile ossicular chain	Using graft
ii. Type II	Perforated tympanic membrane with erosion of malleus handle	Placement of graft on incus or remnant of malleus
iii. Type III (myringostapediopexy or columella tympanoplasty)	Absence of malleus and incus	Placement of graft directly on stapes head
iv. Type IV	Presence of only footplate of stapes and exposure to external ear	Placement of graft between oval and round window (creates narrow middle ear with air pocket around round window)
v. Type V (fenestration operation)	Functioning round window with fixed footplate of stapes	Creation of another window on horizontal semicircular canal

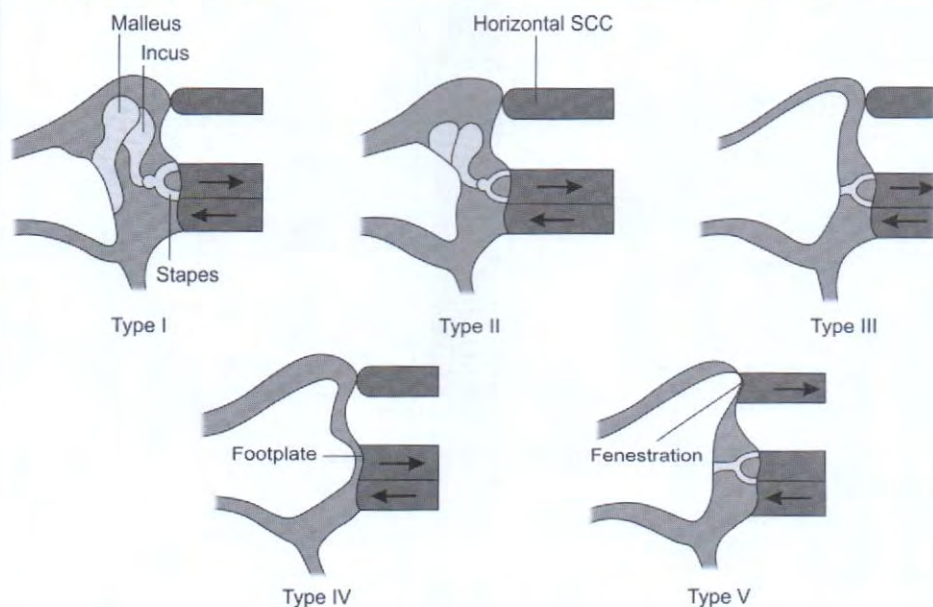


Figure 2: Tympanoplasty—types

Prerequisites for successful tympanoplasty

- Adequate air-bone gap with good cochlear reserve
 - Disease free middle ear and mastoid
 - Properly functioning Eustachian tube (to provide aeration to middle ear).
- c. Myringoplasty (with or without ossicular reconstruction)
- Myringoplasty is closure of tympanic membrane perforation using graft
 - Restores hearing and prevent repeated infection
 - Done after ear becomes dry.

Indications	Contraindications
<ul style="list-style-type: none"> ♦ Tympanic membrane perforation ♦ Hearing loss <40 dB 	<ul style="list-style-type: none"> ♦ Active discharge from middle ear ♦ Nasal allergy ♦ Otitis externa ♦ Ingrowth of squamous epithelium into middle ear ♦ Deaf contralateral ear ♦ Children <3 years

Anesthesia

- Local.

Position

- Supine with face turned such that ear to be operated is superior.

Procedure

Underlay technique	Overlay technique
<ul style="list-style-type: none"> ♦ Incise along edges of perforation and removal ring of epithelium along with strip of mucosal layer from inner side of perforation ♦ Make stapes type incision and raise tympanomeatal flap to visualize integrity and mobility of ossicular chain and to assess any ingrowth of squamous epithelium into middle ear ♦ Pack middle ear with antibiotic soaked gelfoam ♦ Place proper sized graft such that its edges extend under margins of perforation all round and a small part also covers posterior canal wall ♦ Replace tympanomeatal flap 	<ul style="list-style-type: none"> ♦ Incise circumferentially inside meatus and raise meatal skin along with all epithelium from outer surface of tympanic membrane remnant ♦ Place graft on outer surface of tympanic membrane and make a slit in it such that it tucks under handle of malleus ♦ Replace meatal skin, covering periphery of graft ♦ Pack ear canal with gelfoam and antibiotic pack ♦ Close endaural or postaural incision followed by mastoid dressing

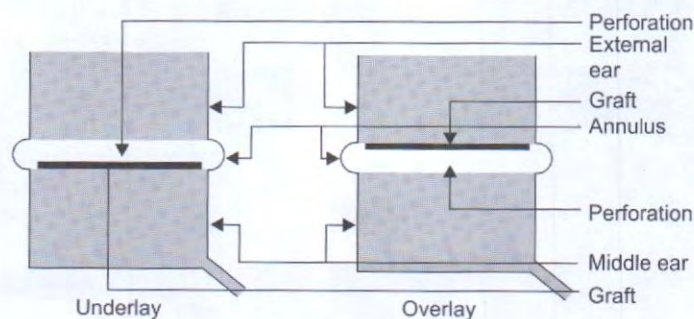


Figure 3: Myringoplasty—types

Postoperative care

- Remove stitches after 5–6 days
- Remove ear pack after 5–6 days
- Follow-up visit at 3rd and 6th week after surgery.

Complications

<i>Underlay technique</i>	<i>Overlay technique</i>
<ul style="list-style-type: none"> ♦ Narrowing of middle ear ♦ Adherence of graft to promontory ♦ Anterior perforation (due to lose of graft contact with remnant of tympanic membrane) 	<ul style="list-style-type: none"> ♦ Blunting of anterior sulcus ♦ Epithelial pearls ♦ Lateralization of graft

2. Discuss in detail the causes and management of epistaxis.

Refer Question No. 1 June 2009 (RS2).

■ SHORT ESSAYS**3. Fibrous dysplasia.**

Refer Question No. 10 December 2009 (RS2).

4. Pure-tone audiometry.

- Pure tone audiometry is a subjective audiological procedure to measure hearing acuity using pure tones both qualitatively and quantitatively.

Principle

- Series of sounds at various frequencies are present to either ear to measure intensity at which sound is heard and degree of deafness.

Procedure

- Performed in soundproof or quite room
- Initially air conduction of both ear is recorded followed by bone conduction
- Sounds are tested in frequency range of 125–8000 Hz
- Usual frequencies tested are 125, 250, 500, 1000, 2000, 4000, 6000 and 8000 Hz
- For each frequency, intensity of sound is gradually increased till subject easily hear it and, then, slowly decreases till subject can no longer hear
- Intensity at which subject hears sound is taken as threshold of hearing at that frequency
- Findings of pure-tone audiometry are plotted on a chart called audiogram as follows:
 - Frequency (in cycles/sec) is plotted on X-axis
 - Hearing capacity (in decibels) is plotted on Y-axis
 - Marking is done as follows:

<i>Marking</i>	<i>Right ear</i>	<i>Left ear</i>
Air conduction	'O'	'X'
Bone conduction	'>'	'<'

Observation

	<i>Conductive deafness</i>	<i>Sensorineural deafness</i>
♦ Bone-conduction threshold	Normal	Raised
♦ Air-conduction threshold	Raised	Raised
♦ Air-bone gap	Present	Absent
♦ Other features	Usually affects lower tones Loss usually <60–70 dB Carhart's notch (otosclerosis)	Dip at 4000 Hz (noise induced hearing loss) Bilateral sloping curve (presbycusis)

Interpretation

Diagnosis	Findings
♦ Normal	Threshold between 0–20 dB
♦ Conductive deafness	Reduced air conduction and normal bone conduction, i.e. air-bone or AB gap
♦ Sensorineural deafness	Reduced air conduction and bone conduction Deafness more severe in higher frequencies
♦ Mixed deafness	Reduced air conduction, partially reduced bone conduction Some gap between air conduction and bone conduction

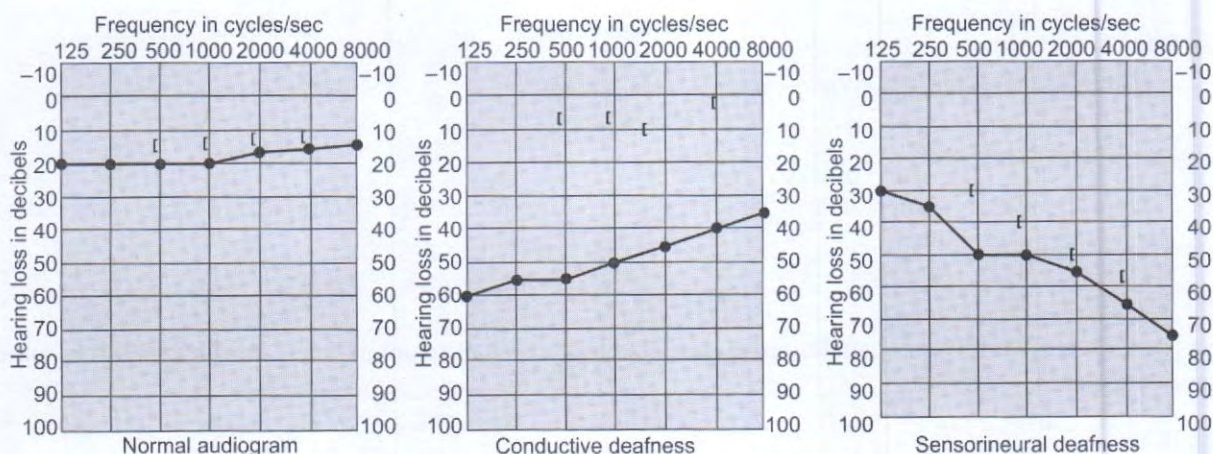


Figure 4: Pure-tone audiometry—findings

Advantages	Disadvantages	Applications														
<ul style="list-style-type: none">♦ Testing of various frequencies♦ Give idea about both type of hearing loss (qualitative) and degree of hearing loss (quantitative)♦ Documentation for future reference♦ Can provide baseline for various reconstructive and rehabilitative procedures to compare serial audiograms before and after treatment♦ Can detect malingers and, thus, helpful in medicolegal cases	<ul style="list-style-type: none">♦ Subjective test♦ Requires patient able to understand instructions♦ Cannot detect malingering♦ Masking needs to be done to avoid involvement of non-test ear	<ul style="list-style-type: none">♦ Measures threshold of hearing by air and bone conduction and, thus, degree of type of hearing loss <table><tr><th>Degree of deafness</th><th>Hearing threshold</th></tr><tr><td>– Mild</td><td>20–30 dB</td></tr><tr><td>– Moderate</td><td>31–60 dB</td></tr><tr><td>– Moderately severe</td><td>61–70 dB</td></tr><tr><td>– Severe</td><td>71–90 dB</td></tr><tr><td>– Profound</td><td>>91 dB</td></tr><tr><td>– Stone (total) deafness</td><td>No hearing</td></tr></table> <ul style="list-style-type: none">♦ Essential for prescription of hearing aid♦ Helps to find degree of handicap for medicolegal purpose♦ Helps to predict speech reception threshold	Degree of deafness	Hearing threshold	– Mild	20–30 dB	– Moderate	31–60 dB	– Moderately severe	61–70 dB	– Severe	71–90 dB	– Profound	>91 dB	– Stone (total) deafness	No hearing
Degree of deafness	Hearing threshold															
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– Moderately severe	61–70 dB															
– Severe	71–90 dB															
– Profound	>91 dB															
– Stone (total) deafness	No hearing															

5. Ludwig's angina.

- Ludwig's angina is infection of submandibular space with cellulitis and occasional abscess formation
- Submandibular space lies between mucus membrane of floor of the mouth and tongue on one side and superficial layer of deep cervical fascia between hyoid bone and mandible on other side and is divided into sublingual and submaxillary and submental spaces by mylohyoid muscle.

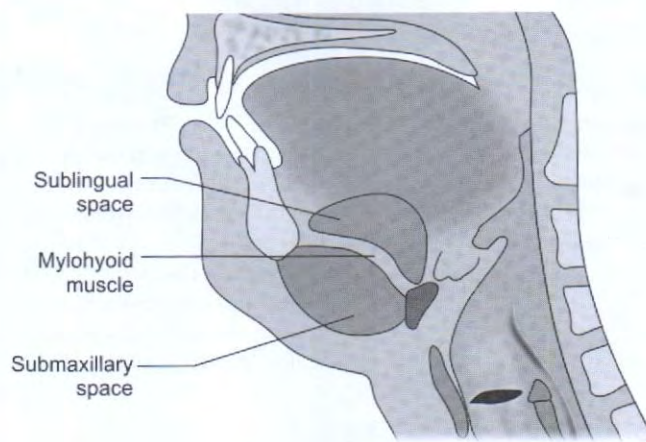


Figure 5: Ludwig's angina

Etiology

<i>Causative agents</i>	<i>Source of infection</i>
<ul style="list-style-type: none"> ♦ Streptococci (alpha hemolytic) ♦ Staphylococci ♦ Bacteroides 	<ul style="list-style-type: none"> ♦ Dental infection (80%), especially of lower 2nd and 3rd molars ♦ Submandibular sialadenitis ♦ Infection or injuries of oral cavity ♦ Lingual tonsillitis ♦ Infection following dental extraction ♦ Fracture of mandible

Clinical Features

- Common in age group of 20–50 years.

<i>Symptoms</i>	<i>Signs</i>
<ul style="list-style-type: none"> ♦ Painful swallowing ♦ Fever ♦ Salivation 	<ul style="list-style-type: none"> ♦ Trismus of varying degree ♦ Swelling of structures in the floor of the mouth ♦ Tongue pushed upward and backward threatening to block airway ♦ Tender brawny swelling of submandibular region with woody hard feel ♦ Stridor due to laryngeal edema

Investigations

- Radiography
 - Dental X-ray to assess dental status
 - CT scan to know extent of disease.

Differential Diagnosis

- Acute submandibular sialadenitis
- Acute cervical lymphadenitis
- Infected plunging ranula
- Infection of necrotic tumor mass
- Tumors or granuloma of submandibular gland.

Treatment

<i>Supportive</i>	<i>Conservative</i>	<i>Operative</i>
<ul style="list-style-type: none"> ♦ Tracheostomy <ul style="list-style-type: none"> – In cases of threatened airway blockage 	<ul style="list-style-type: none"> ♦ Broad spectrum systemic antibiotics along with metronidazole ♦ Anti-inflammatory drugs along with serratiopeptidase 	<ul style="list-style-type: none"> ♦ Incision and drainage <ul style="list-style-type: none"> – Intraoral incision at point of maximum fluctuation (if infection localized to sublingual space) – External transverse incision 2 finger breadth below mandibular margin from one angle of mandible to another over area of induration along with vertical opening of midline musculature of tongue with blunt hemostat (if infection involves submaxillary space) followed by retaining drainage tube or antibiotic soaked ribbon gauze and allowing incision to heal by secondary intention

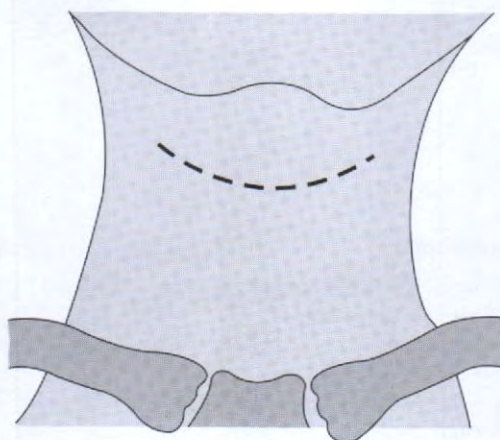


Figure 6: Ludwig's angina—incision

Complications

- Spread of infection to parapharyngeal and retropharyngeal space and thence to mediastinum
- Airway obstruction due to tongue pushed back or laryngeal edema
- Tongue necrosis
- Septicemia
- Aspiration pneumonia.

6. Indications and complications of bronchoscopy.

- Rigid bronchoscopy is endoscopic examination of bronchi using rigid metallic tube (bronchoscope).

<i>Indications</i>			<i>Contraindications</i>
<i>Diagnostic</i>	<i>Therapeutic</i>	<i>Others</i>	
<ul style="list-style-type: none"> ♦ To evaluate cases of wheezing, hemoptysis or unexplained cough >4 weeks ♦ To assess lung if chest X-ray shows <ul style="list-style-type: none"> – Atelectasis of a segment, lobe or entire lobe – Opacity localized to a segment or lobe of lung – Obstructive emphysema—to exclude foreign body – Hilar or mediastinal shadow 	<ul style="list-style-type: none"> ♦ Foreign body removal (Jackson's dictum—in tracheobronchial foreign body, it is safer to perform bronchoscopy rather than withholding endoscopy) ♦ Clearing airway of retained secretions or mucus plug in cases of head injury, chest trauma, thoracic or abdominal surgery or comatose patients 	<ul style="list-style-type: none"> ♦ Prolonged intubation ♦ Difficult intubation ♦ Bronchography ♦ Lobar gas sampling ♦ Placement of endobronchial stents ♦ YAG and CO₂ laser bronchoscopy ♦ Placement of radioactive brachytherapy 	<ul style="list-style-type: none"> ♦ Trismus ♦ Diseases of cervical spine like fracture, caries ♦ Marked kyphosis ♦ Bilateral vocal cord palsy ♦ Vascular tumors like aneurysms of aorta ♦ Hemoptysis ♦ Fulminate suppurative pneumonitis ♦ Moderate or marked dyspnea

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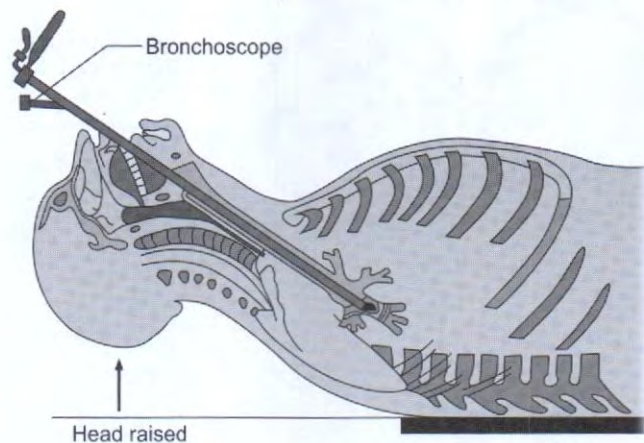
- | | | |
|---|---|--|
| <ul style="list-style-type: none"> ♦ Vocal cord palsy ♦ Collecting bronchial secretions for culture and sensitivity tests, acid-fast bacilli, fungus or malignant cells ♦ Biopsy of endobronchial growth | <ul style="list-style-type: none"> ♦ Dilatation of bronchial stenosis ♦ Lung lavage in asthma, cystic fibrosis and alveolar proteinases | <ul style="list-style-type: none"> ♦ Recent coronary occlusion or cardiac decompensation ♦ Bleeding disorders, hypertension, anemia, diabetes ♦ Cardiac arrhythmias |
|---|---|--|

Anesthesia

- General anesthesia with no endotracheal tube.

Position

- Supine position with elevated head (10–15 cm) by placing pillow under occiput, thus, flexing neck at thorax and extending head at atlanto-occipital joint (Boyce's position or barking-dog position), thus, bringing oral cavity, pharynx and larynx in straight line.

**Figure 7:** Bronchoscopy—position of patient**Procedure**

- Place a piece of gauze on upper teeth to prevent injury to them
- Lubricate a proper sized bronchoscope with autoclaved liquid paraffin or jelly
- Hold bronchoscope with right hand in a pen-like fashion and retract upper lips with left hand to guide bronchoscope into oral cavity through right side of angle of mouth
- Identify tip of epiglottis through scope and pass scope behind it to lift it forward, thus, exposing glottis
- Rotate bronchoscope by 90° clockwise, such that beveled tip is in axis of glottis (tip to right), thus easing its entry into trachea
- Once in trachea, rotate bronchoscope to its original position
- Examine entire tracheobronchial tree while gradually advancing bronchoscope
- To correspond axis of bronchoscope to axis of trachea and bronchi, flex head and neck to contralateral side while examining bronchial tree (i.e. to left when examining right bronchial tree)
- Examine openings of all segmental bronchi in both lungs
- Take biopsy of suspected lesion or collect secretions for investigations
- Slowly retract bronchoscope in reverse fashion as introduced.

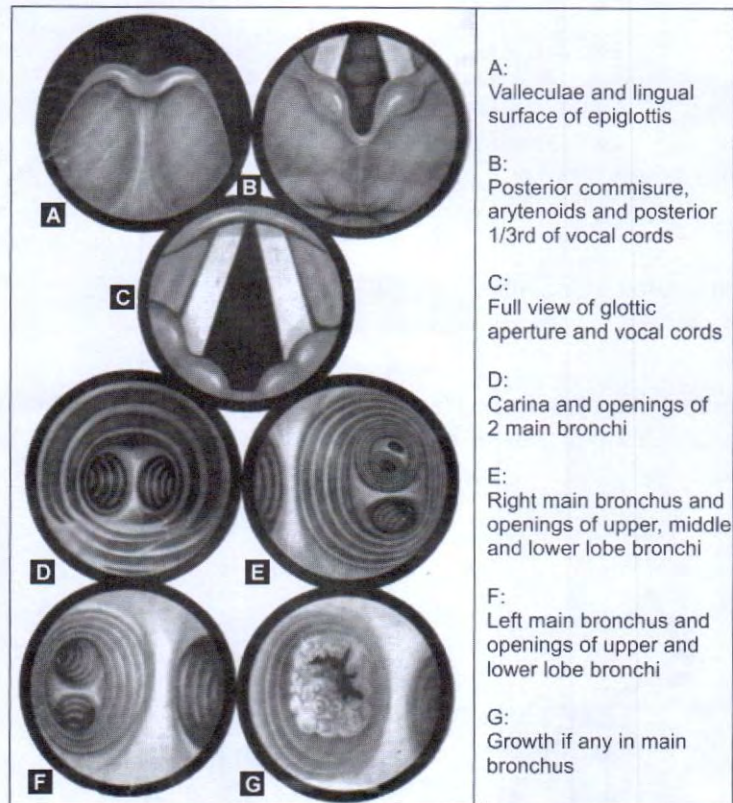


Figure 8: Bronchoscopy—structures seen

Postoperative Care

- Keep patient in humid atmosphere
- Watch for respiratory distress due to laryngeal spasm or subglottic edema if procedure is unduly prolonged or bronchoscope inserted repeatedly.

Complications	Advantages (in comparison to flexible bronchoscopy)	Disadvantages
<ul style="list-style-type: none"> ♦ Injury to teeth, lips, tongue and vocal cords ♦ Hemorrhage from biopsy site ♦ Hypoxia and cardiac arrest ♦ Laryngeal edema particularly of subglottis ♦ Dislocation of cervical vertebrae (if diseased) ♦ Slipping of foreign body or disintegration of foreign body 	<ul style="list-style-type: none"> ♦ Good exposure and airway control ♦ Allows foreign body removal ♦ Dilates strictures 	<ul style="list-style-type: none"> ♦ Requires general anesthesia ♦ Cannot visualize upper lobe and distal segments

7. Clinical features of acoustic neuroma.

- Acoustic neuroma is tumor of VIII cranial nerve
- Also called vestibular schwannoma, neurilemmoma or eighth nerve tumor.

Site of Origin

- From Schwann cells of vestibular division (rarely cochlear division) of VIII nerve at Schwann glial junction near Scarpa's ganglion in internal auditory meatus (Obersteiner-Redlich zone).

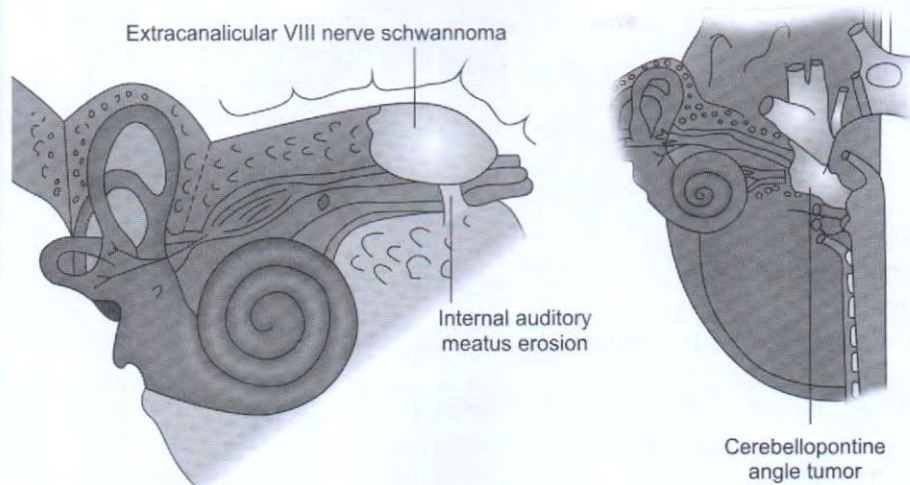


Figure 9: Acoustic neuroma—sites of origin

Spread

- Tumor mostly arises in distal neurilemmal portion of vestibular division of VIII nerve and expands causing widening and erosion of internal auditory canal to appear in cerebellopontine (CP) angle, where it may spread anterosuperiorly to involve V nerve or inferiorly to involve IX, X and XI nerves.

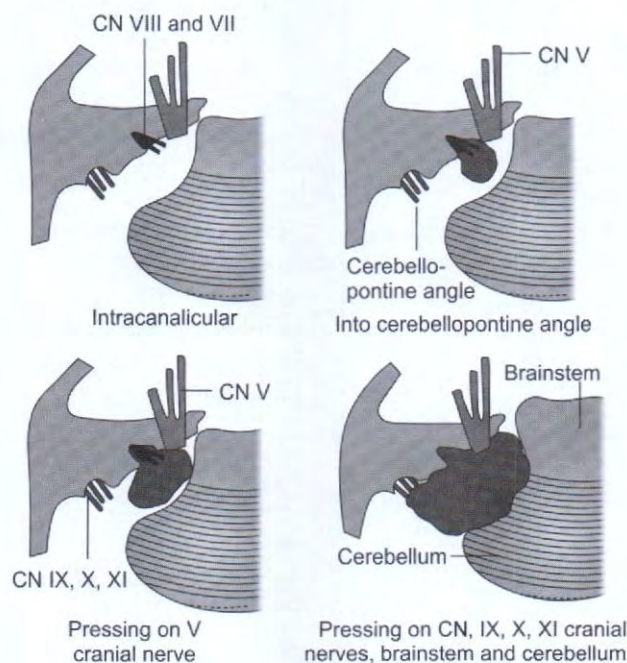


Figure 10: Acoustic neuroma—spread

Pathology

Gross appearance	Microscopic examination
<ul style="list-style-type: none"> ♦ Benign, encapsulated tumor of 8th nerve ♦ Small tumors are pink or yellow and rubbery whereas large tumors are yellow, mottled and cystic 	<ul style="list-style-type: none"> ♦ Shows interlacing bundles of elongated spindle shaped cells with rod shaped nuclei lying in rows or palisades forming ribbon-like fasciculated structures called Verocay bodies (Antoni type A) ♦ Sometimes, there is degeneration and scattering of nuclei in connective tissue fibrils (Antoni type B)

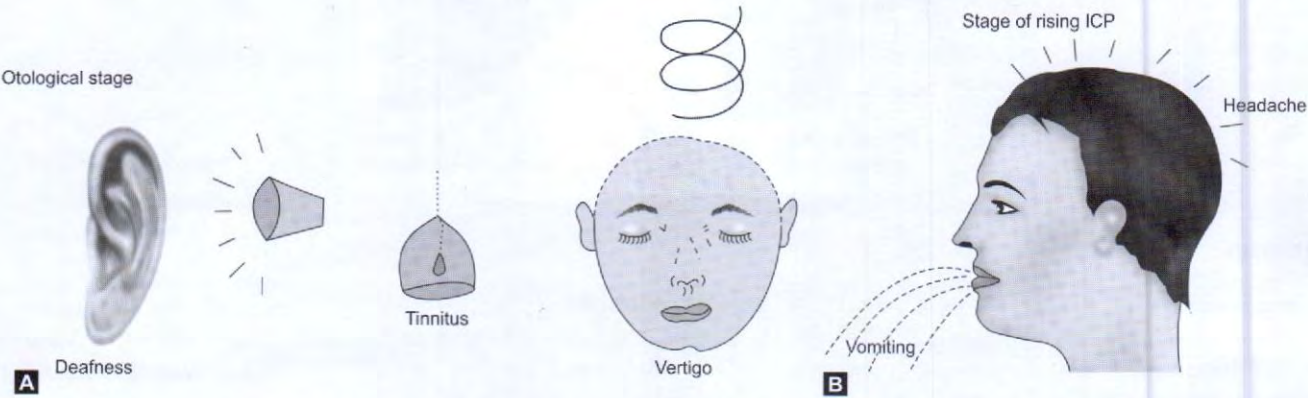
Classification

Intracanalicular	Small size	Medium size	Large
Confined to internal auditory canal	<1.5 cm	1.5–4 cm	>4 cm

Clinical Features

- Extremely slow growing tumor
- Seen mostly in age group of 40–60
- Common in females than males
- Mostly unilateral but bilateral in neurofibromatosis patients.

Symptoms	Signs
a. Cochleovestibular (due to pressure on cochlear or vestibular nerve or internal auditory artery) <ul style="list-style-type: none">– Progressive unilateral hearing loss (sometimes, sudden hearing loss)– Marked difficulty in understanding speech (out of proportion to pure-tone hearing loss)– High pitched continuous tinnitus on affected side– Abrupt onset vertigo	a. Cochleovestibular <ul style="list-style-type: none">– Sensorineural hearing loss (suggest tumor still in internal ear)– Horizontal rotatory nystgmus (Brun's nystagmus)
b. Cranial nerve involvement <ul style="list-style-type: none">– Pain and tingling along distribution of V cranial nerve– Loss of taste– Dysphagia and hoarseness of voice (due to palatal, pharyngeal and laryngeal paralysis)	b. Cranial nerve involvement <ul style="list-style-type: none">– 5th (tumor measures 2.5 cm and is at cerebellopontine angle)– Reduced corneal sensitivity– Numbness or paresthesia of face– 7th nerve– Delayed blink reflex– Infranuclear facial paralysis– Paresthesia of posterior superior meatal wall (Hitzelberger's sign)– Reduced lacrimation (Schirmer's test)– 9th and 10th nerve– Paralysis of palatal, pharyngeal and laryngeal muscles
c. Brainstem involvement <ul style="list-style-type: none">– Weakness and numbness of arms and legs	c. Brainstem involvement <ul style="list-style-type: none">– Ataxia– Exaggerated tendon reflexes
d. Raised intracranial pressure (late feature) <ul style="list-style-type: none">– Headache (in fronto-occipital region of ipsilateral side; worse in morning or precipitated by head movement or exertion)– Nausea– Vomiting– Diplopia (due to involvement of 6th nerve)– Blurring of vision	d. Cerebellar involvement <ul style="list-style-type: none">– Positive cerebellar signs (inability to perform finger-nose test and knee-heel test, dysdiadochokinesia, ataxic gait, etc.) e. Raised intracranial tension <ul style="list-style-type: none">– Papilledema



Figures 11A and B: Acoustic neuroma—autological features and raised intracranial pressure

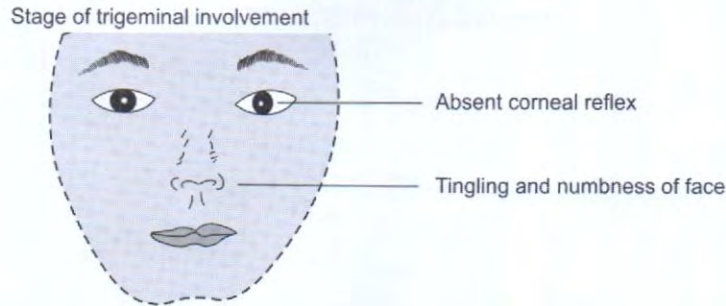


Figure 11C: Acoustic neuroma—trigeminal nerve involvement

Investigations

- a. Audiological tests
 - i. Tuning fork tests
 - Rinne's positive
 - Weber lateralized to affected side
 - Absolute bone conduction decreased.
 - ii. Pure-tone audiometry
 - Unilateral sensorineural hearing loss, more marked in high frequencies.
 - iii. Speech audiometry
 - Poor speech discrimination (disproportionate to pure-tone hearing loss)
 - Rollover phenomenon (reduction of discrimination score when loudness increased beyond particular limit)
 - Absence of loudness recruitment phenomenon.
 - iv. Short increment sensitivity index (SISI) test
 - Show score of 0–20%.
 - v. Threshold tone decay test
 - Tone decay of >30 dB above threshold (retrocochlear type of lesion).
- b. Stapedial reflex decay test
- c. Vestibular tests
 - i. Caloric test
 - Diminished or absent response
 - Normal when tumor is small.
 - ii. Electrocochleography
 - Broadening of AP and S wave.
- d. Radiology
 - Plain X-ray (Towne's view, transorbital AP skull view) shows erosion or widening of internal auditory meatus
 - CT scan detects even smaller ingrowth into posterior cranial fossa and demonstrates difference in diameter of internal auditory meatus
 - MRI with gadolinium contrast detects intracanalicular tumors measuring few mm (gold standard for diagnosis)
 - Vertebral angiography helps to differentiate acoustic neuroma from other CP angle tumors.
- e. BERA
 - Delay of >0.2 msec in wave V between two ears
 - Interval between wave III and wave V >2 msec
 - Interpeak difference between wave I and wave V of >4 msec
 - Absence of wave V for large tumors.
- f. CSF examination
 - Raised protein level.

Differential Diagnosis

- Meniere's disease
- Meningioma
- Primary cholesteatoma
- Arachnoid cyst
- Schwannoma of other cranial nerves.

Treatment

Radiotherapy	Operative
<ul style="list-style-type: none"> ♦ X- or Gamma knife surgery <p><i>Indications</i></p> <ul style="list-style-type: none"> – Surgery contraindicated – Residual tumor <p><i>Procedure</i></p> <ul style="list-style-type: none"> – Form of stereo-tactic radiotherapy involving convergence of radiation energy on tumor, thus causing its growth arrest and reduction in its size without much affecting surrounding normal tissue 	<ul style="list-style-type: none"> ♦ Excision of tumor (treatment of choice) <ul style="list-style-type: none"> – Through various approaches depending upon size <ul style="list-style-type: none"> - Middle cranial fossa approach (for tumor limited to meatus) - Translabyrinthine approach (for tumor limited to meatus) - Suboccipital approach (for large tumors) - Combined translabyrinthine—suboccipital approach

8. Differences between adult and infant's larynx.

Refer Question No. 5 June 2009 (RS2).

9. Adenoid facies.

Refer Question No. 7 June 2016 (RS2).

10. Hearing aids.

- A hearing aid is a device to amplify sound reaching ear by electrical or mechanical means.

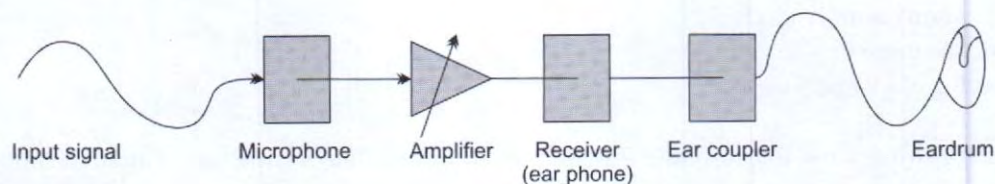
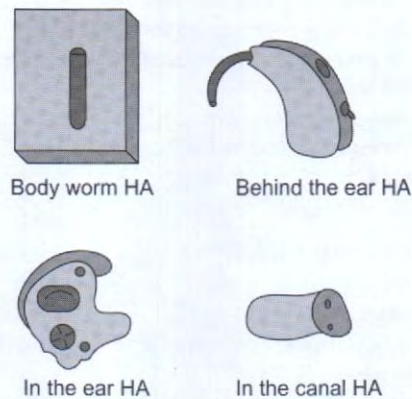
Components

Figure 12: Hearing aid—components

Microphone	Amplifier	Receiver	Batteries
<ul style="list-style-type: none"> ♦ Collects sound and converts it into electrical impulses ♦ Usually of magnetic type ♦ Mechanical hearing aids have microphone in form of diaphragm, which converts sound energy into mechanical energy 	<ul style="list-style-type: none"> ♦ Magnifies electrical impulse by increasing its voltage by implanted transmitter 	<ul style="list-style-type: none"> ♦ Converts electrical impulses back to sound waves ♦ Most are air conduction types ♦ Bone conduction types are also present, which consists of a diaphragm connected to a vibrator 	<ul style="list-style-type: none"> ♦ Used to supply power

Types

a. Bone conduction hearing aid		
b. Air conduction hearing aid	Body worn type	<ul style="list-style-type: none"> ♦ Most common type ♦ Allows high degree of amplification with minimal feedback ♦ Useful in severely deaf and children with congenital deafness
	Behind the ear (BTE) type	<ul style="list-style-type: none"> ♦ A single unit consisting of amplifier, receiver and battery worn behind ear ♦ Coupled to ear canal with tubing and an ear mould ♦ Useful for slight to moderate hearing loss, especially high frequency ones
	Spectacle types	<ul style="list-style-type: none"> ♦ Single unit housed in auricular part of spectacle ♦ Useful for people with hearing defects along with errors of refraction ♦ Not popular now
	In the ear (ITE) type	<ul style="list-style-type: none"> ♦ Entire hearing aid is housed in ear mould which can be worn ♦ Useful for mild-to-moderate hearing loss with fat configuration ♦ Popular because of cosmetic appearance
	Canal types (ITC and CIC)	<ul style="list-style-type: none"> ♦ Very small hearing aid that fits snugly into ear canal ♦ Useful for mild-to-moderate cases of hearing loss to high frequency

**Figure 13:** Hearing aid—types

Indications	Disadvantages
<ul style="list-style-type: none"> ♦ Sensorineural deafness ♦ Conductive deafness ♦ Deaf children ♦ Reluctant patients ♦ Contraindications to surgery 	<ul style="list-style-type: none"> ♦ Hearing aid simplifies to amplify all frequencies within its range, it does not select or emphasize certain frequencies over others like ear ♦ Rarely restores hearing acuity to 100% ♦ Use is only palliative, i.e. it does not cure deafness nor it does arrest disease producing deafness

11. Osteomeatal complex.

Refer Question No. 9 December 2009 (RS2).

12. Differential diagnosis of membrane over tonsil.

- Numerous conditions besides tonsillitis manifest with white patch on tonsils because any lesion in oral cavity becomes white due to slough formed by action of saliva on an open wound.

Differential Diagnosis of White Patch on Tonsil

A. Congenital

- Tonsillar cyst
- A cyst on tonsil appears like a white patch.

B. Traumatic

<i>Traumatic ulcer</i>	<i>Foreign body</i>	<i>Post tonsillectomy wound</i>	<i>Thermal injuries</i>	<i>Radiotherapy</i>
<ul style="list-style-type: none"> ♦ History of trauma due to tooth brush, pencil held in mouth, fingering in throat ♦ White membrane develops within 24 hours 	<ul style="list-style-type: none"> ♦ Foreign body lodged in tonsillar crypt 	<ul style="list-style-type: none"> ♦ History of tonsillectomy ♦ Absence of tonsils 	<ul style="list-style-type: none"> ♦ History of ingestion of hot food or exposure to chemicals 	<ul style="list-style-type: none"> ♦ Seen in cases with head and neck malignancy treated with radiotherapy

C. Infection

Acute		
a. Bacterial		
<i>Membranous tonsillitis</i>	<i>Diphtheria</i>	<i>Vincent's angina</i>
<ul style="list-style-type: none"> ♦ Sudden onset ♦ Fever with chills and rigor ♦ Dysphagia due to severe pain ♦ Exudative membrane over medial surface which can be easily wiped with swab 	<ul style="list-style-type: none"> ♦ Insidious onset ♦ Low or normal temperature ♦ Less local discomfort ♦ Firm, leathery, dirty grey coloured membrane extending beyond tonsil on to soft palate ♦ Strongly adherent and leaves behind bleeding surface when removed ♦ Markedly enlarged lymph nodes ♦ Microbiological studies reveal <i>Corynebacterium diphtheria</i> 	<ul style="list-style-type: none"> ♦ Insidious onset ♦ Less fever and less discomfort ♦ May be unilateral or bilateral ♦ Grayish necrotic membrane covering tonsil or pharyngeal mucosa ♦ Can be easily removed revealing deep irregular ulcer ♦ Microbiological studies reveal fusiform bacilli or spirochetes
b. Viral		
<i>Herpes</i>	<i>Infectious mononucleosis</i>	
<ul style="list-style-type: none"> ♦ Extremely painful ♦ Gray membrane surrounded by red halo ♦ Single or multiple ulcers in row ♦ Heals spontaneously 	<ul style="list-style-type: none"> ♦ Affects young adults ♦ Marked local discomfort ♦ Tonsils enlarged, congested and covered with membrane ♦ Splenomegaly and enlarged lymph nodes in posterior triangle of neck ♦ Failure of antibiotic regime ♦ Lymphocytosis with atypical lymphocytes ♦ Paul-Bunnell test shows high titre of heterophile antibody 	
c. Fungal		
<i>Thrush</i>		
<ul style="list-style-type: none"> ♦ Seen in infants in debilitated people ♦ Milky white, curd-like patches ♦ Discrete, slightly raised ♦ Removable leaving behind inflamed base 		
Chronic		
<i>Tuberculosis</i>	<i>Syphilis</i>	
<ul style="list-style-type: none"> ♦ Severe pain ♦ Secondary to pulmonary infection ♦ Multiple white patches over tonsil 	<ul style="list-style-type: none"> ♦ Snail track ulcer on tonsils, pillars, palate and pharyngeal wall ♦ History of primary chancre 	

D. Blood dyscrasias

<i>Agranulocytosis</i>	<i>Leukemia</i>
<ul style="list-style-type: none"> ♦ Severely ill patient ♦ Ulcerative necrotic lesion in oropharynx besides tonsil ♦ Decreased total leucocyte count (<2000/cu mm) 	<ul style="list-style-type: none"> ♦ Increased total leucocytic count (>100,000/cu mm) ♦ Associated with progressive anemia ♦ Blast cells in bone marrow smear

E. Malignancy

<i>Benign</i>	<i>Malignant</i>
<ul style="list-style-type: none"> ♦ Papilloma presents white patch over tonsil 	<ul style="list-style-type: none"> ♦ Hard irregular white patch with granulations and everted edge ♦ Bleeds readily on touch

F. Miscellaneous

<i>Aphthous ulcer</i>	<i>Tonsillar keratosis</i>	<i>Tonsillolith</i>	<i>Tonsillar cyst</i>
<ul style="list-style-type: none"> ♦ Involve any part of oropharynx ♦ Very painful ♦ Variable size 	<ul style="list-style-type: none"> ♦ Whitish horny outgrowth ♦ Adherent at base ♦ Recurs after removal 	<ul style="list-style-type: none"> ♦ Hard whitish patch projecting from cysts 	<ul style="list-style-type: none"> ♦ Yellowish white colour membrane projecting from cysts ♦ Single or multiple

■ SHORT ANSWERS**13. Otomycosis.**

Refer Question No. 10 June 2009 (RS2).

14. Septal perforation.

Refer Question No. 12 June 2011 (RS2).

15. Tracheostomy tubes.

Refer Question No. 12 June 2016 (RS2).

16. Nerve supply of external auditory canal.

- External acoustic canal is osseocartilagenous canal lined by skin and limited medially by tympanic membrane.

Nerve Supply

Anterior wall and roof	Posterior wall and floor
↓	↓
Auriculotemporal nerve	Auricular branch of vagus (Arnold's nerve) Fibers of facial nerve through vagus

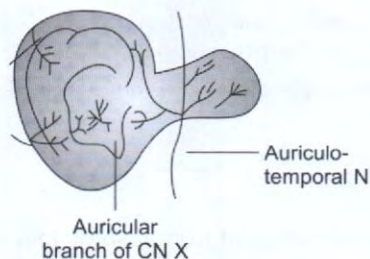


Figure 14: Nerve supply of external auditory canal

Applied Anatomy

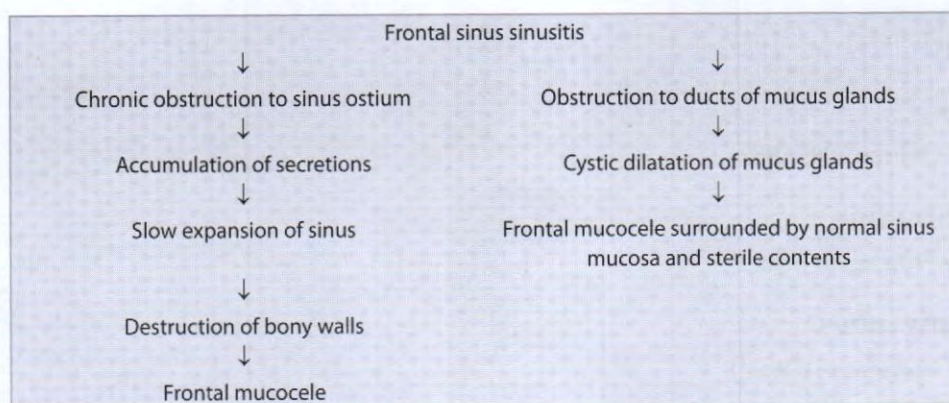
- Stimulation of Arnold's nerve may cause coughing and vasovagal syncope.

17. Frontal mucocele.

- Mucocele is a cystic swelling of sinus secondary to blockage of sinus ostium resulting in thinning and expansion of sinus wall
- Frontal mucocele is a local complication of frontal sinusitis
- Most common mucocele of all paranasal sinuses mucoceles.

Etiology

Etiology	Predisposing factors
♦ Frontal sinusitis	♦ Chronic disease of frontal recess ♦ Postsurgical/traumatic fibrosis

Pathogenesis and Pathology**Clinical Features**

- Presents in superomedial quadrant of orbit (90%).

Symptoms	Signs
♦ Cystic swelling above and lateral to inner canthus of eye and sometimes forehead	♦ Non-tender swelling at superomedial quadrant of orbit
♦ Headache (mild and dull, limited to frontal region)	♦ Swelling is bony initially but later becomes cystic with egg shell cracking
♦ Diplopia (due to proptosis) and ptosis	♦ Proptosis (forward, downward and lateral displacement of eyeball)

Investigations

a. X-ray/CT scan of frontal sinus	♦ Enlarged frontal sinus with clouding and rounding of borders (loss of typical scalloped outline of frontal sinus)
b. Diagnostic nasal endoscopy	♦ Swelling at region of attachment of middle turbinate

Treatment**Operative**

- External frontoethmoidectomy with free drainage of frontal sinus into middle meatus by leaving a drainage tube (Lynch-Howarth's operation)
- Frontal recess clearance and uncapping of mucocele through endoscopic sinus surgery (treatment of choice).

Complications

- Pyocele
 - Due to infection of sterile secretions.

18. Blood supply of palatine tonsil.

Refer Question No. 14 December 2009 (RS2).

19. Ranula.

Refer Question No. 15 June 2011 (RS2).

20. Nasal decongestants.

Refer Question No. 6 December 2008 (RS2).

21. Killian's dehiscence.

Refer Question No. 10 June 2014 (RS2).

22. Carhart's notch.

Refer Question No. 1 December 2010 (RS2).

MBBS PHASE III EXAMINATION

JUNE 2012

(Revised Scheme 2 & 3)

■ LONG ESSAYS

1. Describe briefly the anatomy of the middle ear cleft with a suitable labeled diagram.

- Middle ear cleft or tympanic cavity is an air-filled irregular bony chamber contained within the temporal bone and lies between external auditory meatus and inner ear.

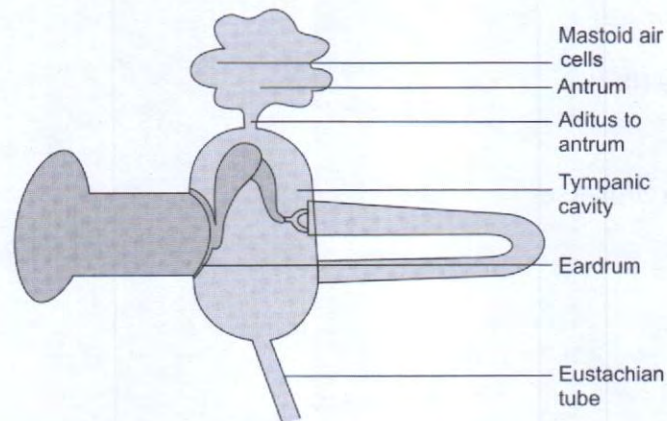


Figure 1: Middle ear cleft

Components

- Tympanum (middle ear cavity proper)
- Eustachian tube
- Mastoid air cell system.

Middle Ear Cavity

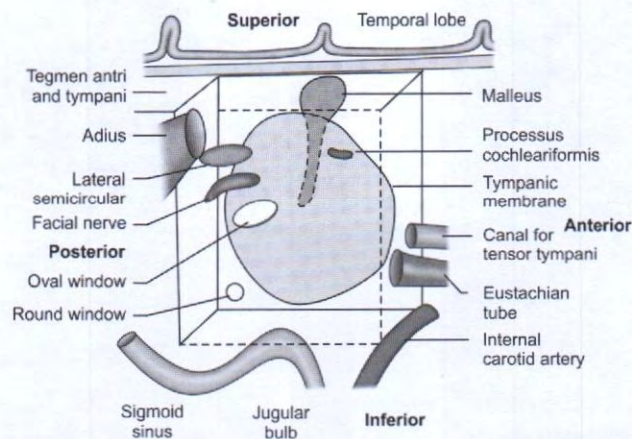


Figure 2: Middle ear cavity

Features**a. Roof (Tegmental wall)**

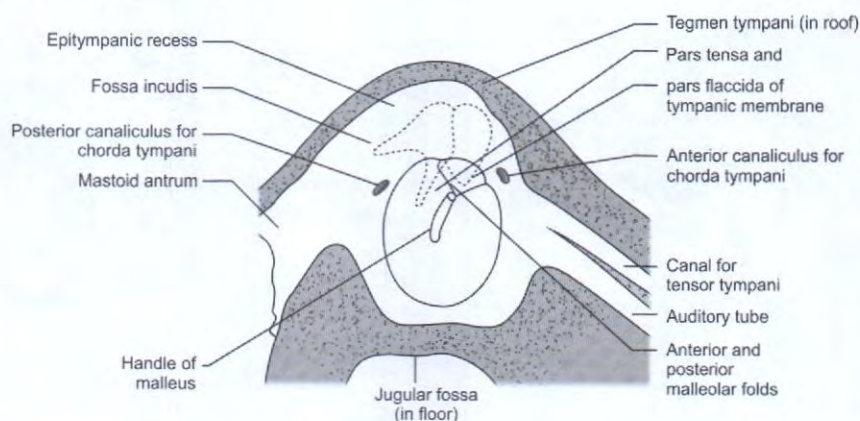
- Formed by tegmen tympani
- Separates middle ear cavity from dura of middle cranial fossa
- Constituted in parts by petrous portion and squamous portion of temporal bone
- Petrosquamous suture line present between these two components is unossified in young, and does not close until adult life and through this suture veins from middle ear may pass to superior petrosal sinus.

b. Floor (Jugular wall)

- Narrower than roof
- Lies in close relationship with jugular bulb but middle ear cavity is separated from jugular bulb by a thin piece of bone (rarely when bone is absent, the separation is only by fibrous tissue and mucous membrane)
- Junction of floor and medial wall presents a small opening which allows entry of tympanic branch of glossopharyngeal nerve which takes an important part in formation of tympanic plexus.

c. Lateral wall (Membranous wall)

- Partly bony and partly membranous

**Figure 3: Lateral wall of middle ear**

- Membranous portion is the central portion formed by tympanic membrane whereas bony portion is above and below tympanic membrane, forming outer lateral walls of epitympanum (attic) and hypotympanum respectively
 - Lateral wall of epitympanum (attic) also includes that part of tympanic membrane lying above anterior and posterior malleolar folds—this portion of eardrum is also known as pars flaccida
 - This portion of tympanic membrane lacks middle fibrous layer, hence the name
 - Lateral attic wall (bony portion) is wedge shaped, its lower portion is also called outer attic wall (scutum)—scutum actually means shield in latin
 - This bony portion is thin and its lateral surface forms superior portion of deep portion of external meatus
 - Three openings are present in bone of medial surface of lateral wall of tympanic cavity
 - i. 1st opening is posterior canaliculus for chorda tympani nerve. This opening is situated at junction between lateral and posterior walls of tympanic cavity. This opening is usually present at level of upper end of handle of malleus. This opening leads to bony canal which descends through posterior wall of tympanic cavity. Since chorda tympani nerve traverses this canal it is also known as canal for chorda tympani nerve. This canal also contains a branch from stylomastoid artery which usually accompanies chorda tympani nerve.
 - ii. 2nd opening is petrotympanic (Glaserian) fissure. This fissure opens anteriorly just above attachment of tympanic membrane. This opening is in fact a small slit about 2 mm long. It receives anterior malleolar ligament. It also transmits anterior tympanic branch of maxillary artery to tympani cavity.
 - iii. 3rd opening is the canal of Huguier. It lies medial to Glaserian fissure. Chorda tympani nerve enters through this.
- d. Anterior wall (Carotid wall)**
- Very narrow because medial and lateral walls converge anteriorly

- Can be divided into two portions; upper and lower
 - i. Lower portion
 - * Larger and has a thin plate of bone which separates this portion from internal carotid artery as it enters the skull
 - * This plate has two openings for caroticotympanic nerves: Upper opening transmits superior caroticotympanic nerve and inferior opening transmits inferior caroticotympanic nerve
 - * It is through these nerves that sympathetic nerves reach the tympanic plexus.
 - ii. Upper portion
 - * Smaller and has two tunnels placed one below the other: Upper tunnel transmits tensor tympani muscle, and lower tunnel transmits bony portion of Eustachian tube.
- e. Medial wall (Labrynthine wall)
 - Separates middle ear from inner ear.

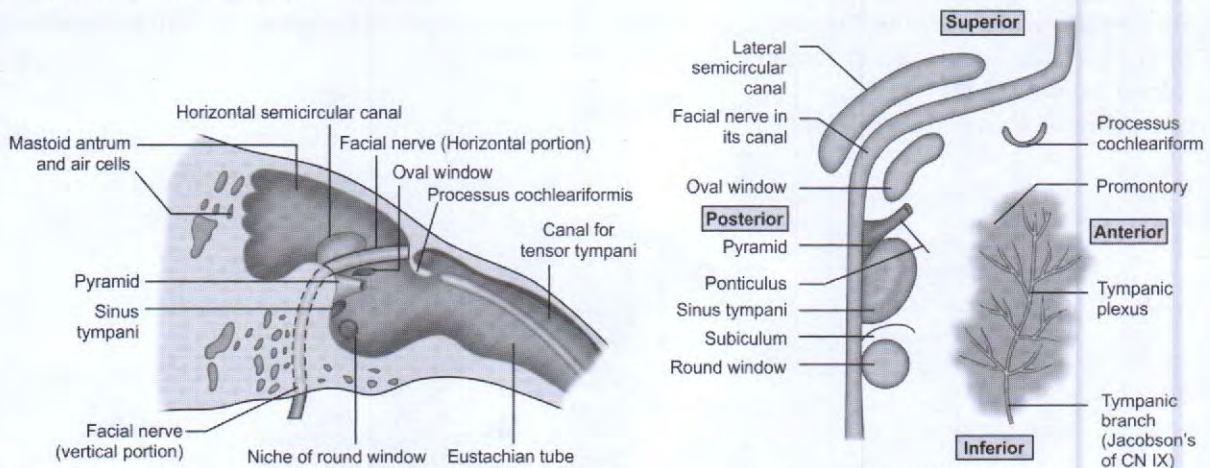
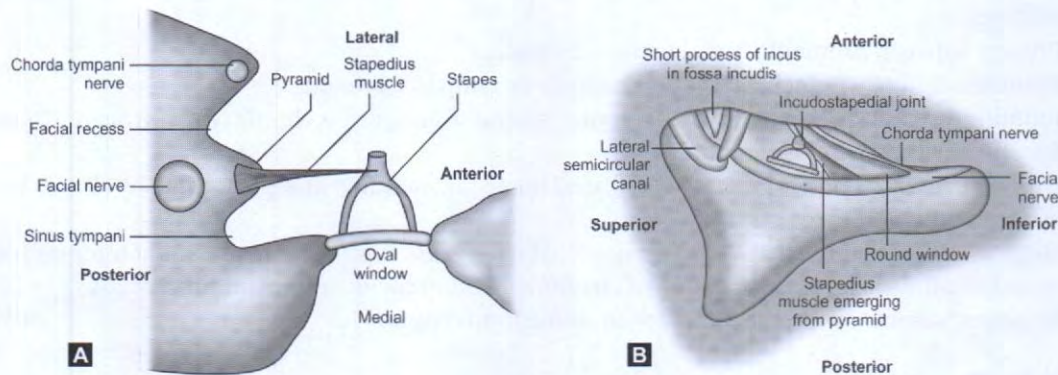


Figure 4: Middle ear—medial wall

- i. Promontory
 - * Most prominent feature
 - * A rounded projection occupying most of central portion of medial wall, raised by underlying basal turn of cochlea
 - * Has numerous small grooves on its surface which contain tympanic plexus of nerves.
- ii. Fenestra vestibuli (oval window)
 - * An oval-shaped opening of variable size but on an average it is 3.25 mm long and 1.75 mm wide
 - * Located behind and above promontory, connecting tympanic cavity with vestibule
 - * Closed by foot plate of stapes and its surrounding annular ligament
 - * Long axis of fenestra vestibuli is horizontal and its inferior border is concave
 - * Above this fenestra vestibuli is canal for facial nerve (horizontal portion) and below lies promontory
 - * Hence, fenestra vestibuli lies at bottom of a depression also known as fossula that can be of varying depths depending on position of facial nerve and prominence of promontory.
- iii. Fenestra cochlea (round window)
 - * Lies just below and behind oval window
 - * Round window faces inferiorly and a little posteriorly, lying completely under cover of promontory and hence usually is difficult to visualize
 - * Round window niche is usually triangular in shape, having anterior, posterosuperior and posteroinferior walls
 - * It is closed by a membrane known as round window membrane (secondary tympanic membrane) of diameter between 1.8 and 2.3 mm
 - * Secondary tympanic membrane is made up of three layers; outer mucosal, middle fibrous and an inner endothelial layer and appears to be divided into an anterior and posterior portions by presence of a transverse thickening

- * Membrane of fenestra cochleae does not lie at end of scala tympani but forms part of its floor and ampulla of posterior semicircular canal is closest vestibular structure to this membrane
 - * Nerve supplying ampulla of posterior semicircular canal (singular nerve) lies close to this secondary tympanic membrane
 - * Secondary tympanic membrane forms a landmark for position of singular nerve
 - * This is useful during surgical procedures like singular neurectomy for treatment of intractable vertigo.
- iv. Subiculum
- * Posterior extension of promontory, which separates two windows (oval and round).
- v. Ponticulus
- * A spicule of bone arising rarely from promontory above subiculum and runs to pyramid on posterior wall of middle ear cavity.
- vi. Sinus tympani
- * Formed by meeting of posterosuperior and posteroinferior walls, posteriorly
 - * Difficult area to visualize and cholesteatoma may lurk in this area making it difficult to remove thus making it the commonest causes of cholesteatoma recurrence after mastoidectomy
 - * Small mirrors known as the zinne mirror can be used to visualize this area indirectly
 - * Since sinus tympani lies under pyramid, removal of pyramid during surgery will bring sinus tympani area into view.



Figures 5A and B: (A) Facial recess and sinus tympani; (B) Structures seen through opening of facial recess

- vii. Canal for facial nerve
- * Another important anatomical structure
 - * Facial nerve runs above promontory and fenestra vestibuli in an anteroposterior direction
 - * Canal may occasionally be deficient leaving an exposed facial nerve and is a dangerous anatomical variant because this nerve can easily be traumatized during any surgical procedures in middle ear cavity
 - * Even infections of middle ear mucosa can cause facial nerve palsy in patients with an exposed facial nerve
 - * Behind fenestra vestibuli, facial nerve turns inferiorly to begin its descent in posterior wall of tympani cavity
 - * Region above level of canal for facial nerve forms medial wall of epitympanum or attic
 - * Dome of lateral semicircular canal extends a little lateral to canal for facial nerve and is major feature of posterior portion of epitympanum. In well-pneumatized bones this dome of lateral canal can be very prominent.
- viii. Processus cochleariformis
- * A bony process at anterior end of facial nerve canal
 - * This curved projection of bone is concave anteriorly and it houses tendon of tensor tympani muscle as it turns laterally to handle of malleus.
- f. Posterior wall (Mastoid wall)
- Wider above than below

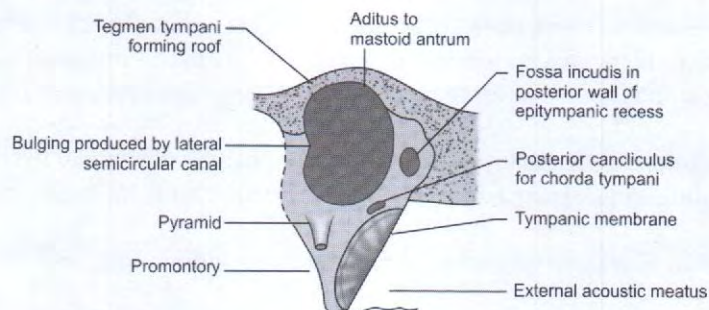


Figure 6: Posterior wall of middle ear

- i. Aditus
 - * A large irregular opening present in upper part, connecting mastoid antrum to middle ear cavity.
- ii. Fossa incudis
 - * A small depression present below aditus, which houses short process of incus.
- iii. Pyramid
 - * A small conical projection located below fossa incudis
 - * It is hollow and its apex pointing anteriorly
 - * It contains stapedius muscle, tendon of which passes forwards to insert into neck of the stapes
 - * Canal within promontory curves downwards and backwards to join descending portion of canal for facial nerve.
- iv. Facial recess
 - * Present between promontory and tympanic annulus
 - * Bounded medially by facial nerve and laterally by tympanic annulus
 - * Running through wall between 2 with varying degree of obliquity is chorda tympani nerve. This nerve always runs medial to tympanic membrane
 - * Drilling over facial recess area between facial nerve and annulus in angle formed by chorda tympani nerve can lead into middle ear cavity
 - * Surgical approach through this area is known as facial recess approach which is suitable for surgeries involving round window niche like placement of electrodes during cochlear implant procedures
 - * Hypotympanum can also be approached through this approach.

Contents of Middle Ear

Contents of middle ear					
Ossicles	Muscles	Nerve fibers	Ligaments	Blood vessels	Air
<ul style="list-style-type: none"> ♦ Malleus ♦ Incus ♦ Stapes 	<ul style="list-style-type: none"> ♦ Tensor tympani ♦ Stapedius 	<ul style="list-style-type: none"> ♦ Chorda tympani nerve ♦ Tympanic plexus of nerve 			Most important content

A. Ossicles

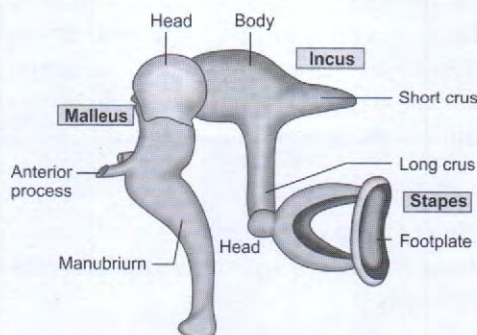


Figure 7: Middle ear

<i>Malleus (hammer)</i>	<i>Incus (anvil)</i>	<i>Stapes (stirrup)</i>
<ul style="list-style-type: none"> ◆ Largest of three ossicles ◆ Hammer shaped bone, hence the name ◆ Has a head, neck and 3 processes arising from below neck ◆ Overall length ranges between 7.5–9 mm ◆ Head lies in attic region, effectively dividing attic into an anterior portion (lying anterior to handle of malleus) and a posterior one (lying behind handle of malleus) ◆ During surgical procedures for attic cholesteatoma, clipping of this head improves exposure in attic region ◆ Posteromedial surface of head of malleus has an elongated saddle-shaped cartilage covered facet for articulation with incus and this articular surface is constricted near its middle dividing articular facet into a larger superior and a smaller inferior portions ◆ Inferior portion of articular facet lies at right angles to that of superior portion and this projecting lower portion is also known as the cog or spur of malleus ◆ Below the neck, bone broadens and gives rise to following <ul style="list-style-type: none"> – Anterior process from which a slender anterior ligament arises to insert into petrotympanic fissure – Lateral process which receives anterior and posterior malleolar folds from annulus tympanicum – Handle which runs downwards, medially and slightly backwards between mucus and fibrous layers of tympanic membrane ◆ Deep medial surface of handle presents a small projection into which tendon of tensor tympani muscle inserts ◆ Additionally malleus is supported by superior ligament which runs from head to tegmen tympani 	<ul style="list-style-type: none"> ◆ Shaped like an anvil and articulates with malleus ◆ Has a body and two processes ◆ Body lies in attic and has a cartilage covered articular facet corresponding to that of malleus ◆ Short process projects backwards from body to lie in fossa incudis and is in fact attached to fossa incudis by a short ligament ◆ Long process of incus descends into mesotympanum behind and medial to handle of malleus ◆ At its tip there is a small medially directed lenticular process which articulates with stapes ◆ Long process of incus has precarious blood supply and thus it is prone for undergoing necrosis in disease conditions 	<ul style="list-style-type: none"> ◆ Consists of a head, neck, two crura and a base (footplate) ◆ Head of stapes points laterally and has a small cartilage covered depression for articulation with lenticular process of incus ◆ Tendon of stapedius muscle attaches to posterior part of neck and upper part of posterior crura ◆ Neck of stapes gives rise to two crura, anterior (thinner and less curved) and posterior crura which join foot plate ◆ Foot plate of stapes closes the oval window ◆ Average dimensions of foot plate is 3 mm × 1.4 mm with its long axis being almost horizontal, with posterior end being slightly lower than anterior

B. Intratympanic muscles

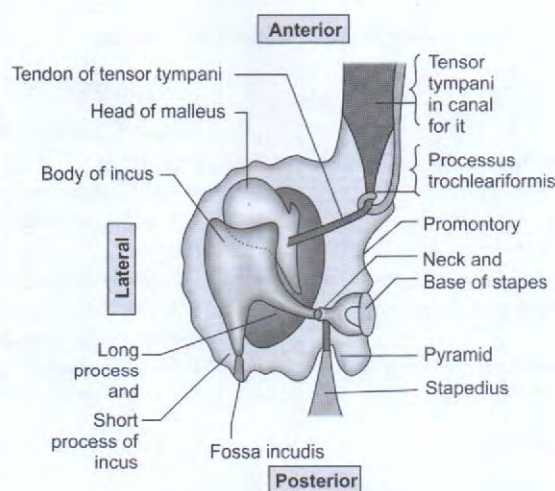


Figure 8: Intratympanic muscles

	<i>Stapedius</i>	<i>Tensor tympani</i>
Location	A small muscle lying in a bony canal, related to posterior wall of middle ear	Lies in a canal that opens into anterior wall of middle ear
Origin	Walls of vertical part of canal of facial nerve	Cartilaginous part of auditory tube Base of skull formed by greater wing of sphenoid Its own bony canal
Course	Fibers end in a tendon that enters middle ear through pyramid and runs forward	Fibers end in a tendon that reaches middle ear cavity near its medial wall where it bends sharply to lateral side by passing around processus trochleariformis
Insertion	Posterior surface of neck of stapes	Upper part of handle of malleus
Nerve supply	Branch of facial nerve	Mandibular nerve (anterior or motor branch)
Action	Contraction usually in response to loud noises Pulls stapes posteriorly and prevents excessive oscillation	Contraction pulls handle of malleus medially, tensing tympanic membrane to reduce force of vibration in response to loud noise
Development	2nd arch	1st arch
Significance	<ul style="list-style-type: none"> Both intratympanic muscles protect ear against very loud sounds by restricting vibrations of tympanic membrane and ossicles Paralysis of muscles (especially stapedius) gives rise to hyperacusis 	

C. Intratympanic nerves

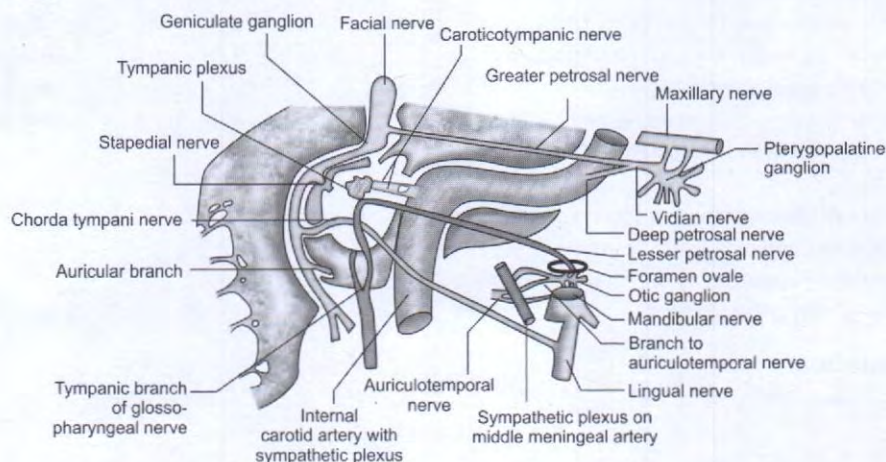


Figure 9: Nerves in relation to middle ear

<i>Chorda tympani nerve</i>	<i>Tympanic plexus</i>
<ul style="list-style-type: none"> A branch of facial nerve It enters middle ear cavity through posterior canaliculus which is present at junction of lateral and posterior walls It runs across medial surface of tympanic membrane between mucosal and fibrous layers passes medial to upper portion of handle of malleus Here it lies above tendon of tensor tympani muscle, continues forwards and leaves by way of anterior canaliculus placed within petrotympanic fissure It joins the lingual branch of V nerve with which it is distributed to anterior 1/3rd of tongue 	<ul style="list-style-type: none"> Found over the promontory <p><i>Formation</i></p> <ul style="list-style-type: none"> Tympanic branch of glossopharyngeal nerve Caroticotympanic nerves (supplies sympathetic component) <p><i>Branches</i></p> <ul style="list-style-type: none"> Branches to mucous membrane lining tympanic cavity, Eustachian tube, mastoid antrum and its air cells A branch joining greater superficial petrosal nerve Lesser superficial petrosal nerve, which contain all parasympathetic fibers of IX nerve

D. Mucosa (varies according to location)

Part	Attic or epitympanum	Middle ear proper	Hypotympanum
Lining epithelium	Pavement epithelium	Cuboidal epithelium	Ciliated columnar epithelium

Functions of Middle Ear

A. Functions of tympanic membrane (TM)

Pressure receiver	Resonator	Critical dampening
TM is extremely sensitive to pressure changes produced by sound waves on its external surface and it acts as a receiver of these pressure changes	TM starts vibrating freely when sound waves strike it thus acting as a resonator	As soon as sound waves stop stretching TM, its vibrations are stopped almost immediately thus it critically dampens sound waves to avoid unnecessary sound additions

B. Function of ossicles

a. Lever system

- Ear ossicles function as a lever system which converts the resonant vibrations of the tympanic membrane into movements of the stapes against the perilymph filled scale vestibuli of the cochlea.

b. Impedance matching (Magnification of sound)

- Some amount of sound energy is lost when a sound wave travels from a thinner medium to denser medium at the interphase of the two mediums.
- Therefore, resistance offered by the denser fluid medium of inner ear to the incoming sound wave leads to loss of about 30 decibels of sound energy at air fluid interphase.
- There are various mechanisms by which the middle ear compensates for this loss and are collectively termed as impedance matching.

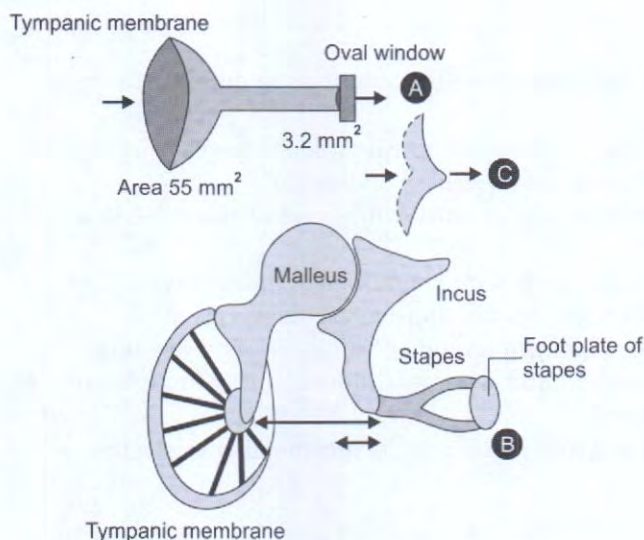


Figure 10: Impedance matching

- These mechanisms are:

Hydraulic action of tympanic membrane	Lever action of ossicles	Buckling factor
<ul style="list-style-type: none"> ♦ Effective vibratory area of tympanic membrane is about 14 times greater than area of oval window thus all force collected over tympanic membrane is concentrated on small oval window ♦ Thus pressure is increased by: $\frac{\text{Area of tympanic membrane}}{\text{Area of oval window}} = \frac{55 \text{ mm}^2}{3.2 \text{ mm}^2} = 17 \text{ times}$ ♦ This is most important factor in achieving impedance matching 	<ul style="list-style-type: none"> ♦ Arm of incus is shorter than handle of malleus and this produces a lever action that increases force and decreases velocity at stapes ♦ This increases pressure by 1.30 times 	<ul style="list-style-type: none"> ♦ This depends on the conical shape of tympanic membrane ♦ As membrane moves in and out, it buckles so that handle of malleus moves less than surface of membrane ♦ This again increases force and decreases velocity ♦ This also increases pressure to some extent

C. Function of muscles

- Tympanic reflex (attenuation reflex)
 - Its main function is to reduce the intensity of loud sounds by 30–40 decibels and protect inner ear

Mechanism

- When loud sound is transmitted through middle ear, a reflex occurs which contracts both stapedius and tensor tympani muscles
- This occurs after a latent period of 40–80 milliseconds
- Inward pull of handle of malleus by tensor tympani and outward pull of stapedius on stapes oppose each other and entire ossicular system develops a kind of rigidity
- This reduces transmission of low frequency sound (loud sounds).

Functions

- This reflex protects cochlea from damaging vibrations caused by excessive loud sounds. However, it can protect the organ of Corti due to latent period of 40–80 milliseconds
- It filter low frequency sounds in a loud environment especially removes major share of background noise thus it is important in voice communications
- It decreases a person's hearing sensitivity to his or her own speech.

Significance

- Thus middle ear plays a very important role in conduction of sound from external ear to inner ear.

Eustachian Tube

- Eustachian tube is bony-cartilagenous tube that connects middle ear cavity with nasopharynx
- It is directed upwards, backwards and outwards from its nasopharyngeal opening and towards its upper opening in anterior walls of middle ear
- Its upper 1/3rd towards middle ear is bony while rest of the tube is a fibrocartilaginous passage
- Nasopharyngeal end of the tube which is on lateral wall of nasopharynx, just behind the posterior end of inferior turbinate normally remains closed
- Tensor palati muscle helps in opening tubal end on swallowing and yawning
- Eustachian tube is short, straight and wide in children and is thought to predispose to middle ear infection
- Nerve supply is derived from tympanic plexus and the sphenopalatine ganglion.

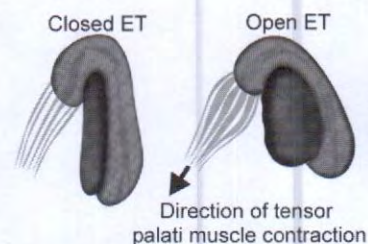


Figure 11: Opening of Eustachian tube by tensor tympani

Mastoid Air Cell System

- It is an air chamber in temporal bone that communicates anteriorly with tympanic cavity through the aditus.

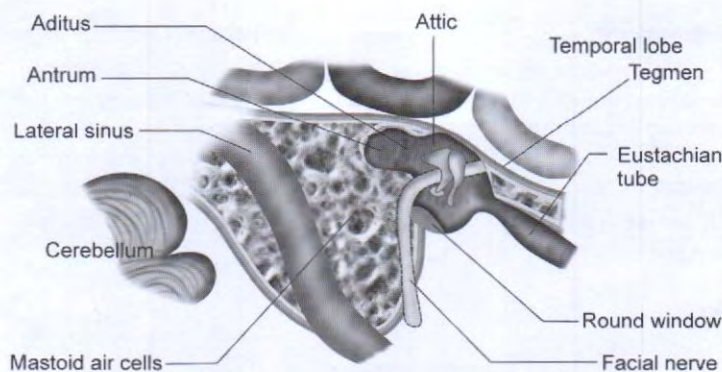


Figure 12: Mastoid air cell system

Features

a. Mastoid antrum

- It lies above and behind projection of a bone called spine of Henle, on posterosuperior angle of canal wall
- Surface anatomy of mastoid antrum is marked by Mac Ewen's triangle
- Cribriform area of bone above and behind this spine is site for antrum which lies about 13 mm deep from surface in adults and only 3 mm deep in infants.

Boundaries

- Medial wall is formed by petrous portion of temporal bone and in this wall lies posterior and lateral semicircular canals
- Lateral wall is formed by squamous portion of temporal bone
- Roof is formed by tegmen antri which separates it from middle cranial fossa
- Posterior wall is formed by mastoid portion of temporal bone
- Floor is formed by mastoid portion of temporal bone.

b. Mastoid process

- It is not present at birth and starts developing at end of 1st year and reaches its adult size at puberty
- It develops posterior to tympanic portion of temporal bone
- In infancy the mastoid process being absent, the facial nerve emerges lateral to tympanic portion from stylomastoid foramen and is likely to get injured by the usual postaural incision.

c. Mastoid air cells

- During development of mastoid process, bone is normally filled with marrow
- Only mastoid antrum and a few periantral cells are present at birth
- With development, mastoid process becomes cellular in a majority of cases (80%) where air cells are large and the intervening septae are thin.

Air cell groups of the mastoid

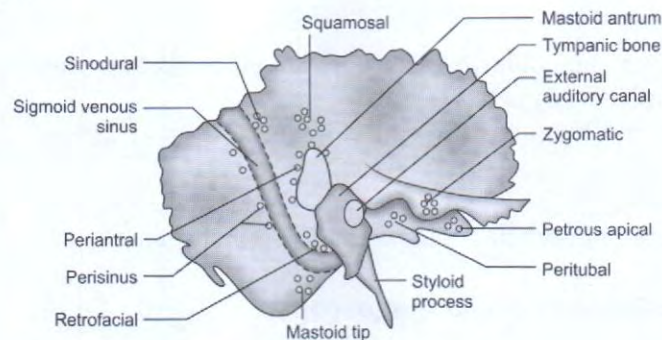


Figure 13: Mastoid air cells

i. Periantral cells	
ii. Tip cells (Superficial and deep tip cells are separated by digastric ridge and facial nerve lies anterior to this ridge)	<ul style="list-style-type: none"> ♦ Large cells lie in the tip of mastoid medial and lateral to digastric ridge <ul style="list-style-type: none"> – Superficial: Lie superficial to posterior belly of digastric muscle – Deep : Lie deep to attachment of posterior belly of digastrics
iii. Perisinus cells	Present over sinus plate
iv. Perilabyrinthine cells	<ul style="list-style-type: none"> ♦ Around labyrinth within petrosa <ul style="list-style-type: none"> – Supralabyrinthine, above arch of superior semicircular canal (may communicate with petrous apex) – Infralabyrinthine, below labyrinth – Retrolabyrinthine, behind labyrinth
v. Retrofacial cells	Present round the fallopian canal of facial nerve
vi. Petrosal cells	Air cells may invade body and apex of petrous bone and may be present under trigeminal ganglion, around internal carotid artery or around Eustachian tube (peritubal cells).

Contd...

Contd...

vii. Marginal cells	Lie behind sinus plate, may extend into occipital bone
viii. Squamous cells	Lie in squamous part of temporal bone
ix. Tegmen cells	In tegmen tympani
x. Hypotympanic cells tracts	
xi. Peritubal	Present around Eustachian tube (These and hypotympanic cells communicate with petrous apex)
xii. Zygomatic cells	In roof of zygoma

2. Classify the causes of epistaxis. Outline the management of epistaxis in a 60-year-old man.

Refer Question No. 1 June 2009 (RS2).

■ SHORT ESSAYS

3. Olfactory area.

- Olfactory area is a specialized area of the nasal mucosa lining the roof of nasal cavity.

Extent

- It is a patch of yellowish pigmented mucous membrane extending laterally to superior concha and medially to upper third of median septum
- It occupies an area of about 2.5 sq cm in each nostril in humans and is covered by mucous produced by Bowman's glands.

Components

- 10–20 million receptor cells interspaced between supporting cells and progenitor cells.

Functions

- During quite breathing, mainstream of inspired air does not reach olfactory area but some air does move up by convection current and reach olfactory area
- However, in sniffing or deep breathing air reaches olfactory area.

Significance

- Olfactory area potential site for transmission of infection to cavernous sinus.

4. Indications and complications of esophagoscopy.

- Rigid esophagoscopy is endoscopic examination of esophagus using rigid metallic tube (esophagoscope)

Indications		Contraindications
Diagnostic	Therapeutic	
<ul style="list-style-type: none"> ♦ Investigate causes dysphagia like cancer, achalasia cardia, strictures, esophagitis, diverticula, etc. ♦ Find cause of retrosternal burning like reflux esophagitis, hiatus hernia ♦ Ascertain causes of hematemesis like esophageal varices ♦ Locate site of tracheoesophageal fistula ♦ Location of unknown primary with secondaries in neck ♦ Unilateral vocal cord palsy 	<ul style="list-style-type: none"> ♦ Foreign body removal ♦ Dilatation of esophageal strictures or achalasia cardia ♦ Endoscopic removal of benign lesions like fibroma, papilloma, cysts, etc. ♦ Endoscopic diathermy of hypopharyngeal pouch (Dohman's operation) ♦ Insertion of Soutar's or Mousseau-Barbin tube in palliative treatment of carcinoma esophagus ♦ Sclerotherapy of esophageal varices ♦ Brachytherapy 	<ul style="list-style-type: none"> ♦ Trismus ♦ Diseases of cervical spine like fracture, caries ♦ Marked kyphosis ♦ Receding mandible ♦ Perforation or necrotic ulceration of esophagus, Mallory-Weiss syndrome ♦ Aneurysms of aorta ♦ Mediastinal growth ♦ Bleeding disorders, hypertension, anemia, diabetes ♦ Recent coronary occlusion or cardiac decompensation ♦ Cardiac arrhythmias ♦ Advanced heart, liver or kidney disease

Anesthesia

- General anesthesia with orotracheal intubation (tube in left corner of mouth).

Position

- Supine position with head projecting beyond table and held elevated (10–15 cm) by assistant thus flexing neck at thorax and extending head at atlanto-occipital joint (Boyce's position or Barking dog position) thus bringing oral cavity, pharynx and esophagus in straight line.

Procedure

- Place a piece of gauze on upper teeth to prevent injury to them
- Lubricate esophagoscope with autoclaved liquid paraffin or jelly
- Hold esophagoscope with right hand in a pen-like fashion and retract upper lips with left hand to guide esophagoscope into oral cavity through right side of angle of mouth
- Move esophagoscope towards middle of dorsum of tongue and advance gently by left thumb and index finger thus visualizing epiglottis, endotracheal tube and arytenoids (in that order)
- Keep tip of esophagoscope in midline, behind larynx in hypopharynx and identify slit like cricopharyngeal opening
- Now apply slow and sustained pressure to open opening and guide tip of esophagoscope into esophagus
- While crossing aortic arch and left bronchus (constriction at 25 cm from incisor), slightly lower head of patient to bring esophageal lumen in line with that of esophagoscope
- Now bring head and shoulder below level of table with head slightly higher than shoulder and slightly tilted to right such that esophagoscope points to left anterior superior iliac spine to visualize cardia
- Now retract esophagoscope in similar fashion while inspecting esophageal wall.

Postoperative care	Complications
<ul style="list-style-type: none"> ♦ Plain water followed by usual diet ♦ Watch for signs of esophageal perforation like pain in interscapular region, surgical emphysema of neck and abrupt rise in temperature 	<ul style="list-style-type: none"> ♦ Injury to lips, teeth, arytenoids, pharyngeal wall ♦ Esophageal perforation, often at Killian's dehiscence due to undue force ♦ Tracheal compression leading to respiratory obstruction and cyanosis ♦ Cardiac arrest ♦ Hemorrhage ♦ Rupture of aortic aneurysm ♦ Dislocation of cervical vertebrae (if diseased)

5. Thyroglossal cyst.

- Thyroglossal cyst is cystic dilatation of remnant of thyroglossal duct
- Thyroglossal duct extends from foramen cecum at base of tongue to isthmus of thyroid passing front of or behind or through body of hyoid bone.

Etiology

Etiology (theories)	Site of cyst
<ul style="list-style-type: none"> ♦ Cystic degeneration of epithelial remnants of thyroglossal duct due to recurrent throat infection ♦ Retention of secretion due to blockin thyroglossal duct 	<ul style="list-style-type: none"> ♦ Subhyoid region (MC)—61% ♦ Suprahyoid region—24% ♦ Infrahyoid region—13% ♦ Beneath foramen cecum—2%

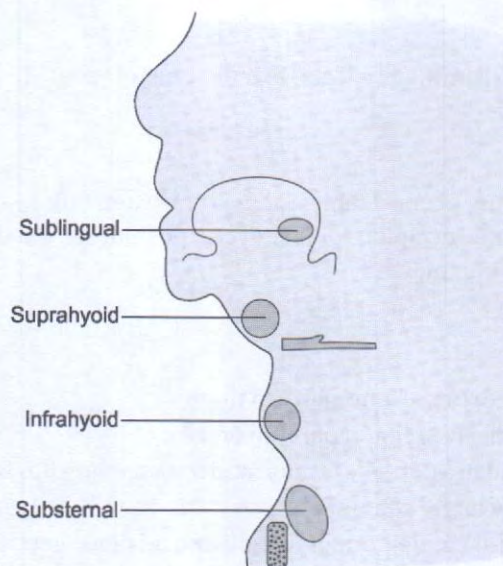


Figure 14: Thyroglossal cyst—sites

Clinical Features

- Usually affects young children under 5 years.

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Swelling in midline of neck below hyoid bone ♦ Painless if not infected ♦ Sudden increases in size following infection ♦ Draining sinus (if burst open due to infection or surgically drained) 	<ul style="list-style-type: none"> ♦ Cystic, rounded swelling measuring 2–4 cm ♦ Moves with deglutition or tongue protrusion (because of its attachment to foramen cecum at base of tongue) ♦ Puckering of skin above fistulous opening (due to pull by thyroglossal tract)—Hood sign

Investigations

Thyroid scan	Fistulogram
<ul style="list-style-type: none"> ♦ To rule out ectopic thyroid tissue 	<ul style="list-style-type: none"> ♦ To identify extent of tract if associated with fistula

Differential Diagnosis

- Dermoid cyst—cheesy secretion
- Lipoma—slippery edges
- Subaceous cyst—doughy feel
- Infected lymph node—purulent secretion
- Thymic cyst
- Subhyoid bursitis
- Hypertrophic pyramidal lobe of thyroid.

Treatment

Operative

- Sistrunk's operation (treatment of choice)
 - Complete surgical excision including with body of hyoid bone, core of tongue tissue around suprahyoid tongue base to foramen cecum.

Complications

- Malignant change (rarely).

6. Organ of Corti.

- Organ Of Corti is the sense organ of hearing as it contains the receptors for hearing, the hair cells
- It is a specialized spiral organ spread like a ribbon along entire length of basilar membrane.

Components

- Hair cells and associated nerve terminals
 - Important receptor cells of hearing and transduce sound energy into electrical energy.
- Supporting cells
 - Deiters' cells are situated between outer hair cells and provide support to latter
 - Cells of Hensen lie outside Deiters' cells.
- Tunnel of Corti
 - Formed by inner and outer rods and contains a fluid called *cortilymph*.
- Tectorial membrane
 - Consists of gelatinous matrix with delicate fibers and overlies organ of Corti
 - Shearing force between hair cells and tectorial membrane produces stimulus to hair cells.

Structure

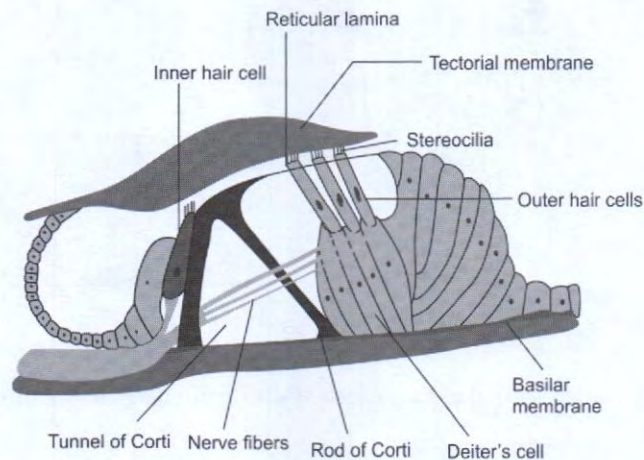


Figure 15: Organ of Corti

- Organ of Corti contains a complex arrangement of auditory sensory cells and supporting cells
- Auditory sensory cells (hair cells) are arranged in two distinct groups—a single row of inner and 3 to 5 rows of outer hair cells
- Body of these hair cells are embedded in organ of Corti while their upper ends contain stereocilia which are free
- Triangular gap between rods of Corti is known as the tunnel of Corti, which contains cortilymph, separates inner and outer hair cells
- Inner hair cells (closer to modiolus) are about 3500 in human cochlea and they form a single row and extend along entire length of cochlea
- Lateral to tunnel of Corti, are three rows of outer hair cells, about 20,000 in number
- Inner hair cells are richly supplied by afferent cochlear fibers and are probably more important in transmission of auditory impulses
- Outer hair cells mainly receive efferent innervation from olivary complex and are concerned with modulating the function of inner hair cells
- Fibers of cochlear nerve run through basilar membrane to hair cells
- Hair cells are surrounded by supporting or sustentacular cells

- Apical ends of hair cells contain cilia (hair) that pass through reticular lamina supported by rods of Corti
- Cilia of outer hair cells project into a thin flexible gelatinous tectorial membrane that covers rows of hair cells, whereas cilia of inner hair cells do not touch the membrane
- Viscous membrane contains collagen and glycoprotein and is firmly attached only along its medial edge.

Function

- When sound waves reach organ of Corti, it acts as a transducer to convert mechanical form of energy into action potentials in cochlear nerve.

7. Gradenigo's syndrome.

Refer Question No. 3 December 2009 (RS2).

8. Dangerous area of the face.

- Dangerous area of face includes upper lip and anteroinferior part of nose including vestibule.

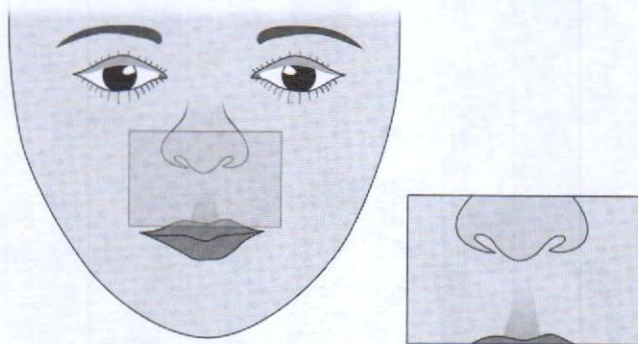


Figure 16: Dangerous area of face

- Any infection in dangerous area can spread to cavernous sinus through following route:
 - Facial vein
 - Pterygoid plexus of veins
 - Ophthalmic veins via frontal and angular veins
 - From middle ear along inferior petrosal sinus
 - From side of head along mastoid emissary veins and superior petrosal sinus
 - From mouth through pterygoid venous plexus.

Significance

- This area freely communicates with cavernous sinus through a set of valveless veins, anterior facial vein and superior ophthalmic vein
- Any infection of this area can thus travel intracranially leading to meningitis and cavernous sinus thrombosis.

9. Functional endoscopic sinus surgery.

Refer Question No. 12 December 2007 (RS2).

10. Etiology and clinical features of vasomotor rhinitis.

- Vasomotor rhinitis is a non-allergic rhinitis characterized by features of nasal allergy.

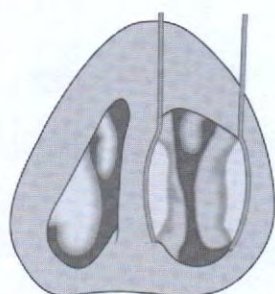
Etiopathogenesis

<i>Etiology</i>	<i>Predisposing factors</i>	<i>Pathogenesis</i>
<ul style="list-style-type: none"> ♦ Heredity ♦ Infection ♦ Psychological ♦ Emotional instability ♦ Endocrine changes (like in pregnancy, puberty, menstruation, etc.) ♦ Drugs (antihypertensives, local decongestants, antidepressants) 	<ul style="list-style-type: none"> ♦ Sudden change in temperature ♦ Humidity ♦ Blasts of air or dust ♦ Alcohol ♦ Stress 	<ul style="list-style-type: none"> ♦ Imbalance of autonomic system, i.e. parasympathetic overactivity causes vasodilatation and engorgement of nasal musosa and also increases secretion from nasal glands

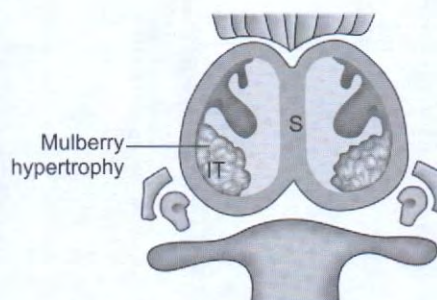
Clinical Features

- Common at younger age especially in women
- Perennial (persists throughout year).

<i>Symptoms</i>	<i>Signs</i>
<ul style="list-style-type: none"> ♦ Paroxysmal sneezing especially just after getting out of bed in morning ♦ Profuse and watery nasal discharge ♦ Nasal obstruction (alternating from side to side) worse at night ♦ Headache and facial pain ♦ Postnasal drip 	<ul style="list-style-type: none"> ♦ Congested and hypertrophied nasal mucosa ♦ Hypertrophied turbinates with mulberry appearance of posterior end of inferior turbinate



Hypertrophied turbinate on anterior rhinoscopy



Mulberry appearance on posterior rhinoscopy

Figure 17: Vasomotor rhinitis—signs**Investigations**

<i>Radiography</i>	<i>Allergic skin tests</i>
<ul style="list-style-type: none"> ♦ X-ray and CT shows hypertrophied mucosa of sinuses 	<ul style="list-style-type: none"> ♦ Negative

Differential Diagnosis

<i>Allergic rhinitis</i>	<i>Infective rhinitis</i>
<ul style="list-style-type: none"> ♦ Seasonal symptoms ♦ Marked sneezing ♦ Bluish nasal mucosa ♦ Positive skin test ♦ Marked eosinophilia ♦ Associated lacrimation 	

Treatment

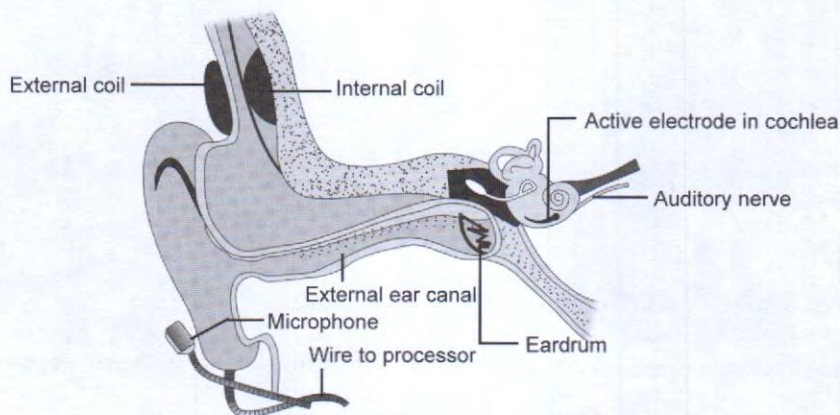
Conservative	Operative
<ul style="list-style-type: none"> ♦ Avoidance or desensitization to predisposing factors ♦ Psychotherapy for emotional imbalance and psychological factors ♦ Antihistaminics ♦ Nasal decongestants ♦ Ipratropium bromide nasal spray to control rhinorrhea ♦ Topical steroids to control symptoms ♦ Systemic steroids for severe cases 	<ul style="list-style-type: none"> ♦ Reduction in size of hypertrophied turbinates to relieve nasal obstruction <ul style="list-style-type: none"> – Submucosal diathermy – Cryotherapy – Bipolar cauterization – Laser turbinoplasty – Partial turbinectomy ♦ Vidian neurectomy to relieve excessive rhinorrhea <ul style="list-style-type: none"> – Sectioning vidian nerve carrying of parasympathetic secretomotor fibers to nose in sphenopalatine fossa through maxillary sinus approach

Complications

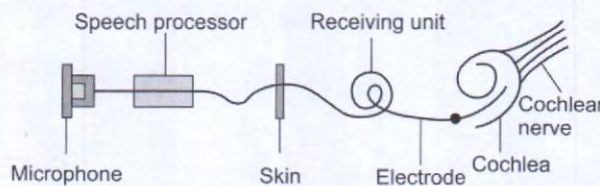
- Nasal polyposis
- Hypertrophic rhinitis
- Sinusitis.

11. Cochlear implants.

- Cochlear implants are electronic devices used in profoundly deaf patients to electrically stimulate cochlear nerve.

**Figure 18: Cochlear implants****Principle**

- Cochlear implants replace nonfunctional transducer system of hair cells of cochlea by converting sound stimulus into electrical stimulus to directly stimulate fibers of 8th cranial nerve.

Components**Figure 19: Cochlear implants—components**

External components (remains outside body)	Internal components (surgically fitted inside body)
a. Microphone – Picks up incoming sound b. Speech processor – Converts sound to digital electric signal – These electrical signals are amplified, compressed and filtered into discrete frequency bands for delivery to appropriate regions of cochlea – Powered by battery and may be body worn or ear level c. Transmitter – Sends signals from external components to internal components by radiofrequency – Held in position by magnet of receiver stimulator	a. Receiver/stimulator – Decodes incoming signals from transmitter – Implanted under skin b. Electrode array – Stimulates fibers of 8th cranial nerve – Consists of multiple electrodes (up to 24 electrodes) and may be single channel or multiple channels – Implanted in scala tympani or over promontory or round window

Examples (Models)

House – 3M device (Los Angeles model)	Nucleus device (Melbourne model)
<ul style="list-style-type: none"> ♦ Single channel intracochlear implant ♦ Electrode is inserted about 6 mm into scala tympani 	<ul style="list-style-type: none"> ♦ Multi-channel intracochlear device ♦ Array of 22 electrodes which are inserted 23 mm into the scala tympani

Prerequisite	Indications	Contraindications
<ul style="list-style-type: none"> ♦ Intact and functional nerve fibers 	<ul style="list-style-type: none"> ♦ Bilateral sensorineural deafness >90 dB hearing threshold ♦ Bilateral cochlear deafness without damage to neural and central connections as in: <ul style="list-style-type: none"> – Presbycusis – Meniere's disease – Ototoxicity – Labyrinthitis – Trauma (surgical or head injury) – Congenital deafness 	<ul style="list-style-type: none"> ♦ Profound deafness caused by meningitis (because meningitis initiates new bone formation leading to cochlear obliteration making insertion of electrodes difficult)

Ideal candidate	Timing
<ul style="list-style-type: none"> ♦ Physically and mentally normal person ♦ Bilateral severe or profound hearing loss ♦ Limited or no benefit from hearing aids ♦ No medical contraindications for surgery ♦ Willingness of patient for postoperative rehabilitation program 	<ul style="list-style-type: none"> ♦ As early as 12 months (earlier the prelingual child undergoes cochlear implant, better the result)

Surgery (Varia Technique)

- Through endaural incision, elevate tympanomeatal flap to expose middle ear and round window
- Make a bed in temporo-occipital bone for receiver stimulator
- Through posterior typanotomy approach, visualize round window
- Perform cochleostomy (widening of round window opening) and drill a passage in bony canal wall from 11 o'clock or 1 o'clock (for right and left ear respectively) to pass electrodes
- Create a seat for receiver/stimulator by elevating posterior part of endaural flap
- Close wound in layers.

Postoperative care	Advantages	Complications	
		Early	Late
<ul style="list-style-type: none"> ♦ Antibiotics and NSAIDs ♦ Remove sutures after 10–12 days ♦ Switch on mapping after 2 months 	<ul style="list-style-type: none"> ♦ No fear of injuring facial canal ♦ Less time consuming ♦ Mastoidectomy is avoided 	<ul style="list-style-type: none"> ♦ Facial paralysis ♦ Wound infection or dehiscence ♦ Flap necrosis ♦ Electrode migration ♦ Device failure ♦ CSF leak ♦ Meningitis ♦ Postoperative dizziness/vertigo 	<ul style="list-style-type: none"> ♦ Exposure of device and extrusion ♦ Pain at implant site ♦ Migration/displacement of device ♦ Late device failure ♦ Otitis media

Follow Up

- Post-implant training program.

Advantages (over hearing aids)	Disadvantages (over hearing aids)
<ul style="list-style-type: none"> ♦ Better efficacy in postlingual deafness ♦ Better results in prelingual deafness ♦ More useful in patients with profound sensorineural hearing loss ♦ Improves speech of prelingual children after intense speech therapy ♦ Improves confidence of patient 	<ul style="list-style-type: none"> ♦ Costly ♦ Cannot be used in psychologically imbalanced individuals ♦ Technically difficult as involves surgical procedure ♦ Patients cannot discriminate words only after intense auditory training ♦ Useful in cases with cochlear hearing loss with normal auditory pathway ♦ Long postoperative rehabilitation ♦ Longer hospital stay

12. Cortical mastoidectomy.

- Cortical mastoidectomy is an exenteration of all accessible mastoid air cells converting them into a single cavity along with preserving posterior meatal wall (middle ear structures are not disturbed)
- Also called as simple or complete or conservative mastoidectomy or Schwartz operation.

Indications

- Acute coalescent mastoiditis
- Acute mastoiditis with impending or coexisting complications
- Incompletely resolved acute otitis media with reservoir sign
- Masked or latent mastoiditis
- As an initial step to perform
 - Endolymphatic sac surgery
 - Decompression of facial nerve
 - Cochlear implantation
 - Labyrinthectomy
 - Cerebrospinal fluid (CSF) otorrhea
 - Access to cerebellopontine angle (acoustic neuroma), skull base, and petrous apex (translabrynthine or retrolabyrinthine procedures for acoustic neuroma).

Position

- Supine with face turned to one side such that ear to be operated is upper most.

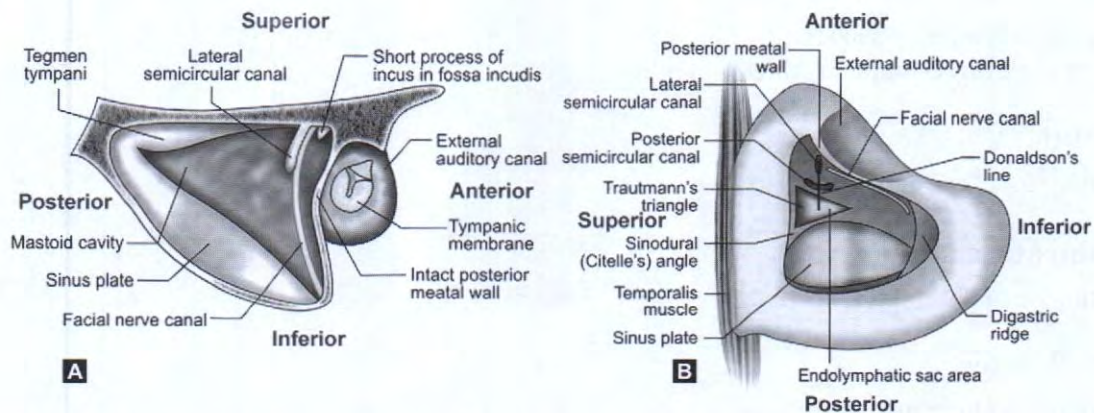
Anesthesia

- General anesthesia.

Procedure

- Incision
 - A curved postaural incision about 1 cm behind but parallel to retroauricular sulcus, starting at highest attachment of pinna to tip of mastoid cutting through soft tissues up to periosteum but preserving temporalis muscle

- Incision is short and more horizontal in children <2 years (to avoid cutting facial nerve which is superficial in lower part of mastoid).
- b. Exposure of mastoid
 - Incise periosteum along line of first incision and scrap it from surface of mastoid and posterosuperior margin of osseous meatus
 - A horizontal incision along lower border of temporalis offers more exposure
 - Cut and scrap down tendinous fibers of sternomastoid
 - Apply a self-retaining mastoid retractor.
- c. Identification of mastoid antrum
 - Drill into mastoid cortex to enter into mastoid antrum which lies 12–15 mm deep to suprameatal triangle (Macewen's triangle)
 - Identify horizontal semicircular canal, aditus ad antrum and short process of incus
 - Remove mastoid antrum with burr, or gouge and hammer.
- d. Removal of mastoid air cells
 - Remove all accessible mastoid air cells (using Lempert's mastoid curette) leaving behind bony plate of tegmen tympani above, sinus plate behind and posterior meatal wall in front.
- e. Removal of mastoid tip
 - Remove lateral wall of mastoid tip, exposing muscle fibers of posterior belly of digastrics
 - Also remove zygomatic cells situated in root of zygoma, retrosinus cells lying between sinus plate and cortex, behind sinus.
- f. Finished cavity
 - Try to identify following structures in the newly created mastoid cavity:
 - *Sinodural angle*: It lies between tegmen antri (middle cranial fossa) and sigmoid sinus
 - *Solid angle*: Solid bone angle, formed by three semicircular canals, lies medial to antrum
 - *Trautmann's triangle*: Bony plate of posterior surface of petrous bone lying behind mastoid antrum; bounded by (i) Sigmoid sinus, (ii) Bony labyrinth and (iii) Superior petrosal sinus
 - *Donaldson's line*: Line passing through lateral semicircular canal bisecting posterior semicircular canal inferior to which lies endolymphatic sac
 - Bevel the edges of finished cavity so that soft tissue can easily sit in and obliterate it.
- g. Wound closure
 - Thoroughly irrigate the mastoid cavity with saline to remove bone dust and close wound in 2 layers
 - Leave behind a rubber drain at lower end of incision for 24–48 hours in case of infection or excessive bleeding
 - Keep a meatal pack to avoid stenosis of ear canal
 - Apply mastoid dressing.



Figures 20A and B: (A) Cortical mastoidectomy with intact posterior meatal wall; (B) Landmarks and structures seen in cortical mastoidectomy cavity

Postoperative Care

- Continue or change antibiotics for at least a week based on antibiotic sensitivity of culture swabs
- Remove the drain after 24–48 hours and perform sterile dressing
- Remove stitches on 6th day.

Complications

- Injury to facial nerve due to heat generated by diamond burr (minimised using constant suction and irrigation)
- Dislocation of incus (results in permanent conductive hearing loss)
- Injury to horizontal semicircular canal (manifests as postoperative giddiness and nystagmus)
- Injury to sigmoid sinus with profuse bleeding or air embolism
- Injury to dura of middle cranial fossa leading to CSF leak
- Postoperative wound infection and wound breakdown
- Bony fixation of malleus and incus.

SHORT ANSWERS**13. Keratosis obturans.**

Refer Question No. 21 June 2008 (RS2).

14. Acoustic trauma.

- Permanent hearing loss due to single brief exposure (as brief as 0.2 ms) to a very intense sound (>140 dB) is termed acoustic trauma.

Etiology

- Sound (impulse noise) could be from explosion, gunfire (most common), powerful cracker which is >140 dB.

Pathogenesis

- Brief and loud noises (140 dB) mechanically damage organ of Corti, tear Reissner's membrane, rupture hair cells, allowing mixing of perilymph and endolymph
- A severe blast (140–170 dB), concomitantly damage tympanic membrane and disrupt ossicles, thus causing additional conductive loss.

Features

- Hearing loss is in range of 4000 Hz
- Does not precede by a temporary threshold shift.

15. Rhinolith.

Refer Question No. 17 December 2008 (RS2).

16. Osteomeatal complex.

Refer Question No. 9 December 2009 (RS2).

17. Adenoid facies.

Refer Question No. 7 June 2016 (RS2).

18. Globus hystericus.

- A psychosomatic disorder
- Also called globus pharyngeus.

Etiology

- Psychological
- Cancer phobia
- Hyperacidity.

Clinical Features

- Usually affects adults and mostly females.

<i>Symptoms</i>	<i>Signs</i>
<ul style="list-style-type: none"> ♦ Lump in throat between meals rather than during a meal ♦ No dysphagia to solids or liquids ♦ Anxiety due to fear of throat cancer 	<ul style="list-style-type: none"> ♦ Normal pharynx, larynx and base of tongue

Investigations

<i>Radiological</i>	<i>Oesophageal</i>
<ul style="list-style-type: none"> ♦ Barium swallow under fluoroscopy ♦ X-ray neck 	<ul style="list-style-type: none"> ♦ Flexible esophagoscopy ♦ Manometric studies

Differential Diagnosis

- Cricopharyngeal spasm
- Pharyngeal pouch
- Gastroesophageal reflux disease
- Achalasia cardia
- Hiatus hernia
- Treatment
- Reassurance
- Tranquillisers to control symptoms
- Placebo treatment
- Psychiatric treatment if required.

19. Lingual thyroid.

Refer Question No. 6 June 2008 (RS2).

20. Stridor.

Refer Question No. 1 June 2016 (RS2).

21. Nasal myiasis.

Refer Question No. 17 December 2008 (RS2).

22. Bezold's abscess.

Refer Question No. 9 June 2008 (RS2).

MBBS PHASE III EXAMINATION

DECEMBER 2012

(Revised Scheme 2 & 3)

LONG ESSAYS

1. Describe the etiology, clinical features and management of acute otitis media.

- Acute suppurative otitis media is an acute inflammation of middle ear cavity by pyogenic organisms.

Etiology

Causative agents	Route of infection	Predisposing factors (anything interfering with normal functioning of Eustachian tube)
<ul style="list-style-type: none"> <i>Streptococcus pneumoniae</i> (30%) <i>Haemophilus influenzae</i> (20%) <i>Moraxella catarrhalis</i> (12%) <i>Streptococcus pyogenes</i> <i>Staphylococcus aureus</i> <i>Pseudomonas aeruginosa</i> 	<p>a. Via Eustachian tube (MC)</p> <ul style="list-style-type: none"> Shorter, wider and horizontal tube in children predispose to spread of infection Horizontal position of infant while breast-feeding or bottle feeding may force fluid through tube into middle ear Swimming and diving also forces water into middle ear through tube Habit of forceful blowing of nose also forces infection through tube <p>b. Via external ear</p> <ul style="list-style-type: none"> In cases of traumatic perforations of tympanic membrane (TM) <p>c. Blood borne</p> <ul style="list-style-type: none"> Uncommon route 	<ul style="list-style-type: none"> Recurrent attacks of cold, upper respiratory tract infections and exanthematous fevers like measles, diphtheria, whooping cough Tonsillitis or adenoiditis Chronic rhinitis and sinusitis Tumors of nasopharynx Packing of nose or nasopharynx (for epistaxis) Cleft palate Barotrauma during flying or diving Low socioeconomic conditions

Pathophysiology (Course)

Stage of tubal occlusion (congestion)	Stage of pre-suppurative (exudation)	Stage of suppuration	Stage of resolution	Stage of complications
<ul style="list-style-type: none"> Blocking of Eustachian tube by edema and hyperemia of nasopharyngeal end of tube Absorption of air in middle ear → -ve intratympanic pressure Retraction of TM with some degree of middle ear effusion 	<ul style="list-style-type: none"> Invasion of middle ear cavity by microorganism causing hyperemia Appearance of mucoid inflammatory exudates Congestion of TM 	<ul style="list-style-type: none"> Formation of pus in middle ear and mastoid air cells Bulging of TM to the point of rupture 	<ul style="list-style-type: none"> Rupture of TM releasing pus Resolving of inflammatory process Relieving of symptoms 	<ul style="list-style-type: none"> In cases of infection with highly virulent organism or poor pt. resistance Infection spread through aditus into mastoid antrum causing catarrhal mastoiditis (congestion of mastoid mucosa) → coalescent mastoiditis (coalescence of mastoid air cells due to breaking of septa under tension by pus) → empyema of mastoid (mastoid full of pus and granulations)

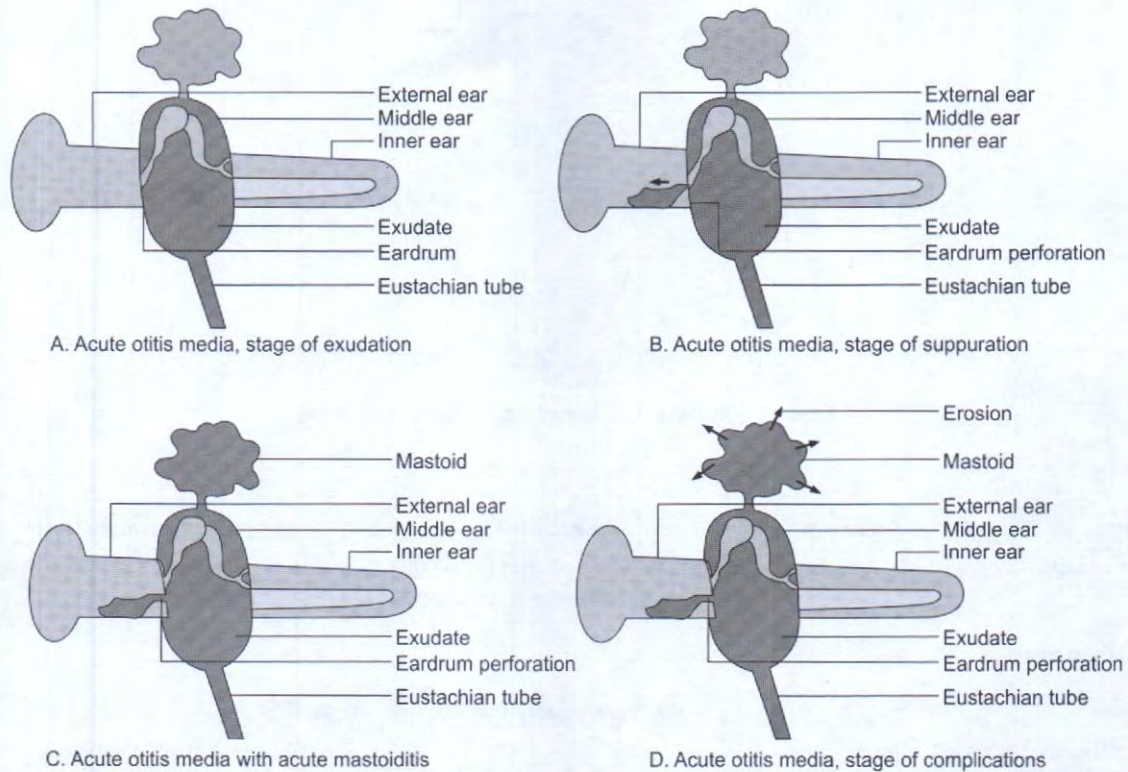


Figure 1: Acute otitis media—stages

Clinical Features

- Commonly affects infants and young children
- Usually follows attack of upper respiratory tract infection.

Symptoms

a. Fever	<ul style="list-style-type: none"> ♦ High degree of fever (102–103°F) making child restless ♦ Associated with vomiting and convulsions
b. Earache	<ul style="list-style-type: none"> ♦ Throbbing in nature ♦ Excruciating ♦ Disturbs sleeps because more at night in recumbent position (due to venous congestion)
c. Deafness	<ul style="list-style-type: none"> ♦ Conductive type of deafness ♦ Increases progressively
d. Fullness in ear	<ul style="list-style-type: none"> ♦ Seen initially
e. Tinnitus	<ul style="list-style-type: none"> ♦ Bubbling sound ♦ Complained only by adults
f. Autophony	<ul style="list-style-type: none"> ♦ Echoing of spoken words in ear

Signs

Stage of tubal occlusion (congestion)	Stage of pre-suppurative (exudation)	Stage of suppuration	Stage of resolution
<ul style="list-style-type: none"> ♦ Retracted TM with handle of malleus assuming more horizontal position ♦ Prominence of lateral process of malleus ♦ Loss of light reflex 	<ul style="list-style-type: none"> ♦ Congestion of pars tensa ♦ Cart wheel appearance of TM (by appearance of leash of blood vessels along handle of malleus and at periphery of TM) ♦ Uniformly reddish TM including pars flaccida 	<ul style="list-style-type: none"> ♦ Reddish TM appears to be bulging with loss of landmarks ♦ Handle of malleus engulfed by swollen and protruding TM ♦ Yellow spot on TM indicating site of imminent rupture ♦ Tenderness over suprameatal triangle (mastoid antrum) 	<ul style="list-style-type: none"> ♦ Blood tinged discharge which later becomes mucopurulent ♦ Small perforation in antero-inferior quadrant of pars tensa ♦ Pulsatile discharge synchronized with each arterial dilatation caused by heart beat (Lighthouse sign) ♦ Resolution of hyperemia of TM

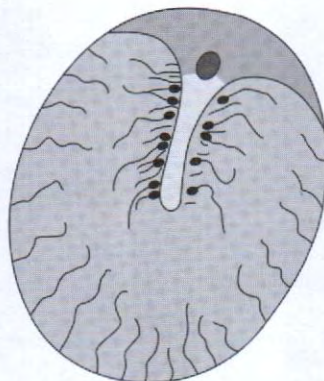


Figure 2: Cart wheel appearance of tympanic membrane

Investigations

<i>Tuning fork tests</i>	<i>Audiometry</i>	<i>X-ray mastoid</i>	<i>Culture and sensitivity of pus</i>
♦ Reveals conductive deafness	♦ Reveals conductive deafness	♦ Clouding of air cells (due to exudates) in stage of suppuration	♦ Reveals causative organism

Differential Diagnosis

<i>Acute otitis media</i>	<i>Herpetic lesions of external ear</i>	<i>Crying child</i>
♦ History of trauma, scratching or water entry ♦ Tenderness below or anterior to pinna ♦ Normal TM		♦ Reddish TM but not edematous ♦ Light reflex present

Treatment*Supportive*

a. Fomentation	♦ To relieve pain ♦ By application of dry heat or instillation of warm oil
b. Ear toilet	♦ Dry mopping of ear discharge with sterile cotton bud followed by insertion of antibiotic moistened wick
c. Analgesics and antipyretics	♦ Paracetamol to relieve pain and fever ♦ Glycerin carbolic ear drops reduce ear pain by destroying superficial nerve endings on external surface of TM
d. Nasal decongestants	♦ To relieve Eustachian tube edema and promote ventilation of middle ear <i>Drugs and dosage</i> ♦ 1% ephedrine nasal drops (0.5% children) ♦ 30 mg pseudoephedrine BD

*Specific**Conservative*

- Antibiotics

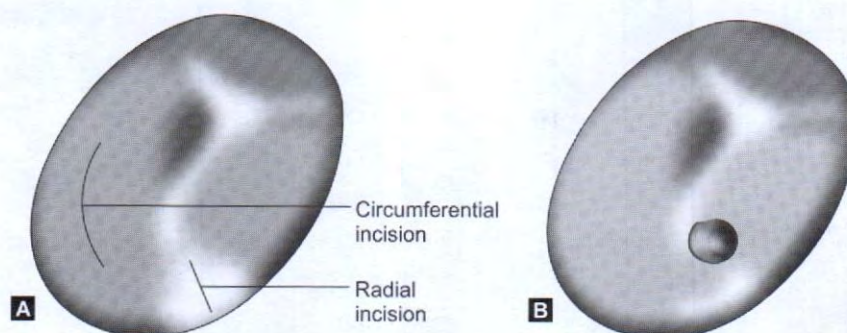
Indications

- All cases with fever and severe earache.

Drugs and dosage

<i>Drug</i>	<i>Dosage</i>		<i>Specific indication</i>
♦ Amoxicillin	40 mg/kg/day	TID	Drugs of choice
♦ Ampicillin	50 mg/kg/day	QID	

Contd...



Figures 3A and B: (A) Myringotomy—incisions; (B) Grommet in tympanic membrane

Postoperative care	Complications
<ul style="list-style-type: none"> ♦ Daily mopping of ear discharge for acute suppurative otitis media ♦ Leave behind a wad of cotton for 24–48 hours for serous otitis media ♦ Antibiotics, analgesics, decongestants ♦ Avoid water entry into ear canal for at least 1 week (or for entire duration of grommet insertion) ♦ Grommet left in place for weeks or months (usually gets spontaneously extruded after 6–8 months) 	<ul style="list-style-type: none"> ♦ Injury to incudostapedial joint or stapes (esp. during circumferential incision) ♦ Injury to posterior meatal wall (due to loss of demarcation between drum head and posterior meatal wall because of inflammation and edema) ♦ Injury to jugular bulb with profuse bleeding (in case of high jugular bulb and dehiscent middle ear floor) ♦ Injury to chorda tympani nerve ♦ Middle ear infection

Complications

- Acute mastoiditis
- Subperiosteal abscess
- Facial paralysis
- Labyrinthitis
- Petrositis
- Extradural abscess
- Meningitis
- Brain abscess
- Lateral sinus thrombophlebitis.

2. Describe the etiology, clinical features and management of ethmoidal polyps.

Refer Question No. 2 December 2013 (RS2).

SHORT ESSAYS

3. Cerebrospinal fluid rhinorrhea.

Refer Question No. 6 June 2010 (RS2).

4. Bell's palsy.

- Bell's palsy is idiopathic, peripheral (infranuclear) facial paralysis of acute onset.

Etiopathogenesis

Etiology	Pathogenesis
a. Idiopathic (in most cases) b. Viral infection – Herpes simplex (HSV-1) – Herpes zoster – Epstein Barr virus c. Vascular ischemia of facial nerve vasculature near stylomastoid foramen – Induced by cold or emotional stress d. Heredity – Hereditary narrowing of fallopian canal predisposes nerve to early compression with slight edema e. Autoimmune disorder	<pre> Etiological factor ↓ Primary ischemia ↓ ↑ capillary permeability ↓ Exudation of fluid ↓ Edema ↓ Compression of microcirculation of nerve ↓ Secondary ischemia </pre>

Clinical Features

- Peak incidence in 3rd and 4th decade of life
- Usually occurs at night during sleep
- Sudden onset and unilateral.

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Inability to close eye on affected side ♦ Dribbling of saliva from angle of mouth ♦ Loss of voluntary movements of muscles of facial expression on affected side ♦ Epiphora (due to defective drainage of tears because of weakness of lower lid) ♦ Pain in ear (before and after onset of paralysis) ♦ Noise intolerance (due to stapedial paralysis) ♦ Loss of taste (involvement of chorda tympani) 	<ul style="list-style-type: none"> ♦ Facial asymmetry ♦ Inability to wrinkle forehead on affected side ♦ Deviation of angle of mouth to normal side ♦ Inability to blow whistle (due to paralysis of buccinator muscle) ♦ Bell's phenomenon (rolling up of eyeball while attempting to close eye)

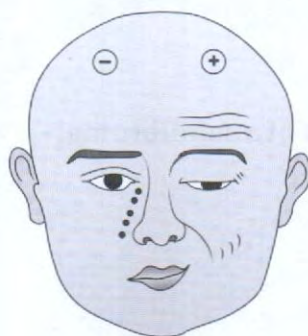


Figure 4: Bell's palsy

Investigations

Blood investigations	Nerve excitability tests	Radiograph of mastoid
<ul style="list-style-type: none"> ♦ Total count, differential count, ESR ♦ Peripheral smear ♦ Blood sugar 	<ul style="list-style-type: none"> ♦ To locate level of lesion and degree of degeneration 	

Treatment*Supportive*

- Reassurance
- Analgesics to relieve pain
- Eye care (padding of eyes or wearing goggles to protect cornea)
- Physiotherapy or massage of facial muscles.

*Specific**Conservative*

Steroids	Antivirals	Vitamins	Vasodilators
Prednisolone (DOC) ♦ 1 mg/kg/day in 2 divided doses for 5 days <i>Follow-up after 5 days to assess progress</i> ♦ Paralysis incomplete or recovering – Tapering dose for next 5 days (total 10 days) ♦ Paralysis complete – Continue for next 10 days followed by tapering dose for next 5 days (total 20 days)	Acyclovir ♦ 800 mg orally or 200 mg IV every 5 hours for 10 days for herpes zoster palsy	Vitamin B1, B6 and B12	Nicotinic acid ♦ Help by relieving vasospasm

Operative

- Nerve decompression

Principle	Indications	Procedure
♦ Relieves pressure on nerve and improve microcirculation	♦ Non recovering Bell's paralysis in 6–8 weeks ♦ Post-traumatic facial palsy	♦ Decompression of vertical and tympanic segment via mastoid and middle ear approach (middle cranial fossa approach for total nerve decompression)

Prognosis

- Complete recovery in 85–90%
- Incomplete recovery in 10–15%
- Recurrence in 3–10%.

5. Sleep apnea syndrome.

Refer Question No. 10 June 2010 (RS2).

6. Clinical features and management of angiofibroma.

Refer Question No. 1 December 2009 (RS2).

7. Complications of tracheostomy.

Refer Question No. 9 December 2007 (RS2).

8. Differential diagnosis of a midline neck swelling.**Midline Swellings of Neck Include (from above downward)***Submental region (2)*

- Submental lymphadenitis.

Between menton and hyoid (3)

- Ludwig's angina
- Sublingual dermoid/midline dermoid

- Ranula/plunging ranula
- Thyroglossal cyst.

Between hyoid and thyroid cartilage (4)

- Subhyoid bursitis
- Osteoma of hyoid bone
- Chondritis/perichondritis
- Chondromas of thyroid cartilage
- Laryngocele.

Between thyroid and cricoid (5)

- Delphian node enlargement.

Between cricoid and suprasternal notch (6)

- Thyroid gland—goiters.

At suprasternal space of burns (7)

- Cold abscess/lymph nodes
- Ectopic thyroid
- Suprasternal bursitis
- Neurofibroma of supraclavicular nerve
- Aneurysm of arch of aorta.

General Rules Applicable to Neck Swellings

A. Rule of 7s for duration of swelling

- If 7 days: Inflammatory
- If 7 months: Neoplastic
- If 7 years: Congenital.

B. 80:20 rule for malignant vs benign neck swellings

- In pediatric age group: 20% are malignant and 80% are benign
- In adult age group: 20% are benign and 80% are malignant.

C. 20:40 rule for age group

<20 years:

- Congenital lesions: Thyroglossal cyst, midline dermoid cyst, branchial cyst, cystic hygroma
- Inflammatory lymph nodes: Tonsillitis, adenoids
- Chronic infections: Tuberculous lymph nodes
- Malignant lesions: Lymphoma, metastatic lymph node (rarely).

20–40 years:

- Salivary gland pathology: Calculus, infections, tumors
- Thyroid pathology: Goiter, neoplasms, thyroiditis, lymphoma
- Chronic infections: Tuberculosis, HIV.

>40 years:

- Primary malignant tumor
- Metastatic lymph node.

9. Nasal bone fractures.

Refer Question No. 3 December 2010 (RS2).

10. Clinical features and treatment of Meniere's disease.

Refer Question No. 2 December 2008 (RS2).

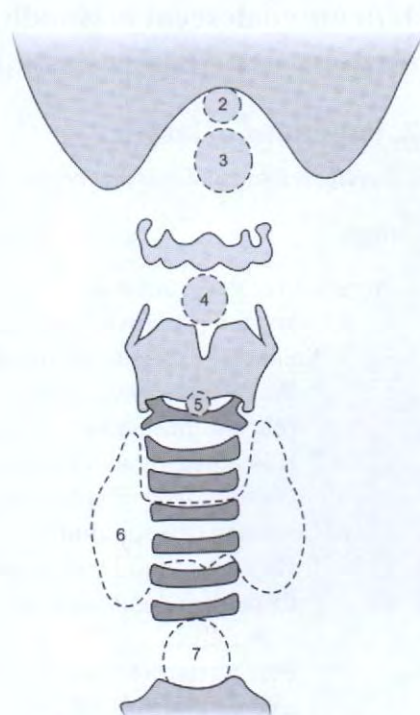


Figure 5: Midline swellings of neck—differential diagnosis

11. Acute coalescent mastoiditis.

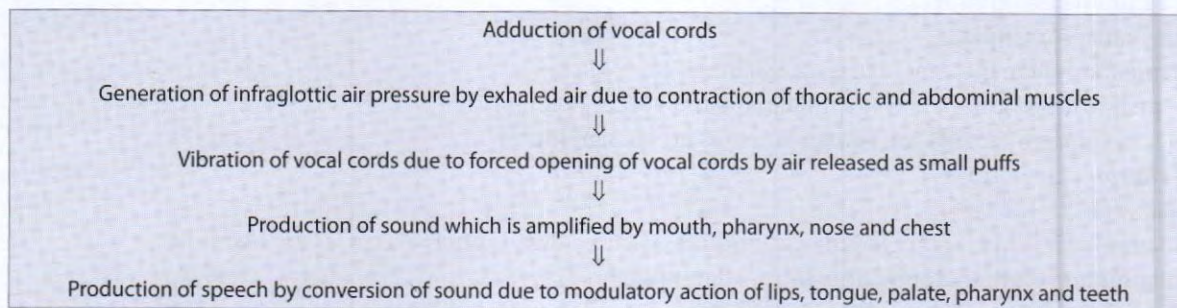
Refer Question No. 3 December 2009 (RS2).

12. Functions of larynx.

- Larynx is a musculocartilagenous skeleton connecting pharynx to trachea.

Functions

- a. Protection of lower airways
 - Primary and primitive function of larynx is protection of lower airways which is achieved as follows:
 - i. Sphincteric closure of laryngeal opening
 - * When food is swallowed, three successive sphincters, i.e. laryngeal inlet, false cords and true cords close reflexly, thus preventing entry of food into larynx
 - * It is aided by elevation of larynx (mylohyoid), tilting of larynx (stylopharyngeous) and relaxation of cricopharyngeal sphincter.
 - ii. Cessation of respiration
 - * Contact of food with base of tongue or posterior pharyngeal wall reflexly cease respiration temporarily through afferent fibers of IX nerve.
 - iii. Cough reflex
 - * Foreign particles which accidentally enter respiratory system (larynx) are dislodged and expelled out through a very powerful and protective cough reflex.
- b. Phonation (aerodynamic myoelastic theory)
 - In higher animals, larynx is so evolved that it acts like a wind instrument which produces voice by following mechanism.



- c. Respiration
 - Larynx regulate entry of air into lungs by reflexly adjusting abduction (during inspiration) or adduction (during expiration) of vocal cords.
- d. Stabilization of thorax
 - With larynx closed, intrathoracic pressure increases making thorax fixed, thus letting all thoracic and abdominal muscles act best, which is an important feature of various physiological processes like coughing, vomiting, defecation, micturition and parturition besides other strenuous activities like climbing, etc.

■ SHORT ANSWERS**13. Rhinitis caseosa.**

- A rare chronic inflammation of nose characterized by formation of granulation tissue and cheesy epithelial debris in nose
- Also called nasal cholesteatoma.

Etiopathogenesis

<i>Etiology</i>	<i>Pathogenesis</i>
♦ As sequel to chronic sinusitis, nasal foreign body or disintegration of nasal polypi	♦ Inspissation of sinus secretions

Clinical Features

- Usually unilateral
- Affects mostly males.

<i>Symptoms</i>	<i>Signs</i>
♦ Unpleasant smell from nose ♦ Offensive purulent discharge	♦ Nose filled with offensive purulent discharge and cheesy material ♦ Granulation tissue in nose along with whitish debris ♦ Destruction of bony wall of sinus

Investigations

- ♦ Histopathology
 - Chronic inflammation of nasal mucosa
 - Debris show keratinous material, cholesterol crystals and numerous organisms.

Differential Diagnosis

- Carcinoma of paranasal sinuses.

Treatment

<i>Conservative</i>	<i>Operative</i>
♦ Removal of debris and granulation tissue	♦ Free drainage of affected sinus

14. Tinnitus.

Refer Question No. 6 June 2015 (RS2).

15. Vestibulitis of nose.

- Vestibulitis of nose is diffuse dermatitis of external nose.

Etiopathogenesis

<i>Causative organism</i>	<i>Predisposing factors</i>	<i>Pathogenesis</i>
♦ <i>S. aureus</i>	♦ Nasal discharge due to rhinitis, sinusitis or nasal allergy ♦ Frequent picking or wiping of nose ♦ Traumatic perforation of projected skin over a caudally dislocated septum	♦ Persistent infected nasal discharge from nose leading to irritation and maceration of skin of vestibule

Types

- Acute and chronic.

Clinical Features

<i>Symptoms</i>	<i>Signs</i>
♦ Red, painful and tender swelling ♦ Itching and irritation of nose (more common in acute form) ♦ Pain in nose	♦ Induration over skin of vestibule (cardinal feature) ♦ Crusts and scales ♦ Excoriation and painful fissures over affected area (common in acute form) ♦ Infection may involve upper lip

Investigations

- WBC count—total and differential count
- X-ray PNS.

Treatment

- Treat cause of nasal discharge
- Clear nasal discharge using suction
- Clean all crusts and scales with cotton soaked in hydrogen peroxide and apply antibiotic-steroid ointment (continue few days after cure to prevent relapse)
- Apply petroleum jelly to form protective barrier
- Cauterize chronic fissure with silver nitrate.

16. Otitis externa.

Refer Question No. 15 December 2008 (RS2).

17. Vocal cord polyps.

Refer Question No. 22 June 2008 (RS2).

18. Cold abscess of neck.

- Cold abscess is a collection of pus and tubercular debris
- It is called a cold abscess because it is not associated with the usual signs of inflammation like heat, redness, etc. as found in pyogenic abscesses
- It may be located anywhere around the vertebrae and are named like wise, i.e. prevertebral, paravertebral, etc.

Etiology (in neck)

- Tuberculous cervical spine (most common)
- Tuberculosis of cervical lymph nodes.

Clinical Features

- Fullness and fluctuant swelling in the neck
- Symptoms of compression due to abscess.

Investigations

Radiology	Microbiology
<ul style="list-style-type: none"> • X-ray neck shows large radiopaque shadow • Chest X-ray to rule out pulmonary tuberculosis 	<ul style="list-style-type: none"> • AFB staining of non-dependent aspiration of cold abscess

Treatment

Conservative	Operative						
<ul style="list-style-type: none"> • Antitubercular regimen as follows: <table border="1"> <tr> <td>Initial 3 months</td><td>Rifampicin + isoniazid + pyrazinamide + ethambutol</td></tr> <tr> <td>Next 9 months</td><td>Rifampicin + isoniazid + pyrazinamide</td></tr> <tr> <td>Next 6 months</td><td>Rifampicin + isoniazid</td></tr> </table> 	Initial 3 months	Rifampicin + isoniazid + pyrazinamide + ethambutol	Next 9 months	Rifampicin + isoniazid + pyrazinamide	Next 6 months	Rifampicin + isoniazid	<ol style="list-style-type: none"> Aspiration <ul style="list-style-type: none"> – Insert a thick bore needle to enable non-dependent aspiration Minimal debridement <ul style="list-style-type: none"> – Evacuate contents of cold abscess and thoroughly curette walls, followed by bone grafting if necessary
Initial 3 months	Rifampicin + isoniazid + pyrazinamide + ethambutol						
Next 9 months	Rifampicin + isoniazid + pyrazinamide						
Next 6 months	Rifampicin + isoniazid						

Complications

- Rupture

<i>Anterior rupture (ruptures deep to prevertebral layer of deep cervical fascia)</i>	<i>Posterior rupture</i>
<ul style="list-style-type: none"> ♦ Pus travels to: <ul style="list-style-type: none"> – Upper cervical region to present as deep seated abscess in posterior wall of pharynx in midline – Lower cervical region pressing on esophagus and trachea – Posterior triangle behind carotid sheath deep to prevertebral fascia 	<ul style="list-style-type: none"> ♦ Pus may enter spinal canal and, then, travel along anterior primary division of cervical spinal nerves

19. Waldeyer's ring.

Refer Question No. 3 June. 2015 (RS2).

20. Septal abscess.

Refer Question No. 8 December 2015 (RS2).

21. Parapharyngeal space.

Refer Question No. 2 December 2009 (RS2).

22. Rinne's test.

Refer Question No. 18 December 2007 (RS2).

MBBS PHASE III EXAMINATION

JUNE 2013

(Revised Scheme 2 & 3)

LONG ESSAYS

1. Describe etiology, clinical features and management of cholesteatoma.

- Cholesteatoma is a sac lined by keratinizing stratified squamous epithelium containing keratinized desquamated epithelium debris in middle ear or mastoid resting on a fibrous tissue layer, i.e. it is skin in wrong place
- Cholesteatoma (cholesteat = cholesterol crystals, oma = tumor) is misnomer because it neither contains cholesterol crystals nor it is a tumor
- Also called epidermosis or keratoma.

Etiology (Genesis/Origin)—Theories

a. Congenital theory	♦ Presence of congenital cell nests, trapped in parietal bone or elsewhere in skull
b. Wittmaack's theory (Retraction pockets)	<ul style="list-style-type: none"> ♦ Eustachian tube malfunction forms retraction pockets in attic or posterosuperior quadrant of eardrum which lines wall of middle ear with help stratified squamous epithelium on outer surface of eardrum ♦ This retraction pockets hamper normal migratory action of epithelium of external auditory meatus resulting in accumulation of desquamated epithelium in attic ♦ This causes pressure necrosis of tympanic membrane forming cholesteatoma of middle ear
c. Ruedi's theory (Basal cell hyperplasia)	♦ Laying down of keratinizing squamous epithelium by basal cells of germinal layer of skin under influence of infection
d. Habermann's theory (Epithelial in growth or theory of migration)	♦ Invasion of epithelium from external canal or outer surface of tympanic membrane through a preexisting perforation (especially marginal type) with destroyed annulus tympanicus or due to absence of middle fibrous layer of drum in attic
e. Sade's theory (Metaplasia)	♦ Metaplasia of middle ear mucosa into squamous epithelium due to repeated infections
f. Implantation theory	♦ Due to implantation of squamous epithelium in middle ear during surgery

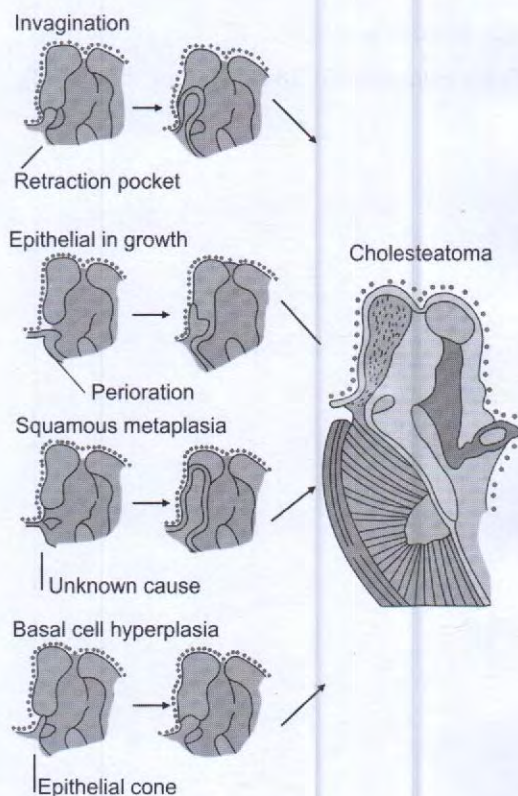


Figure 1: Cholesteatoma—theories of origin

Pathogenesis

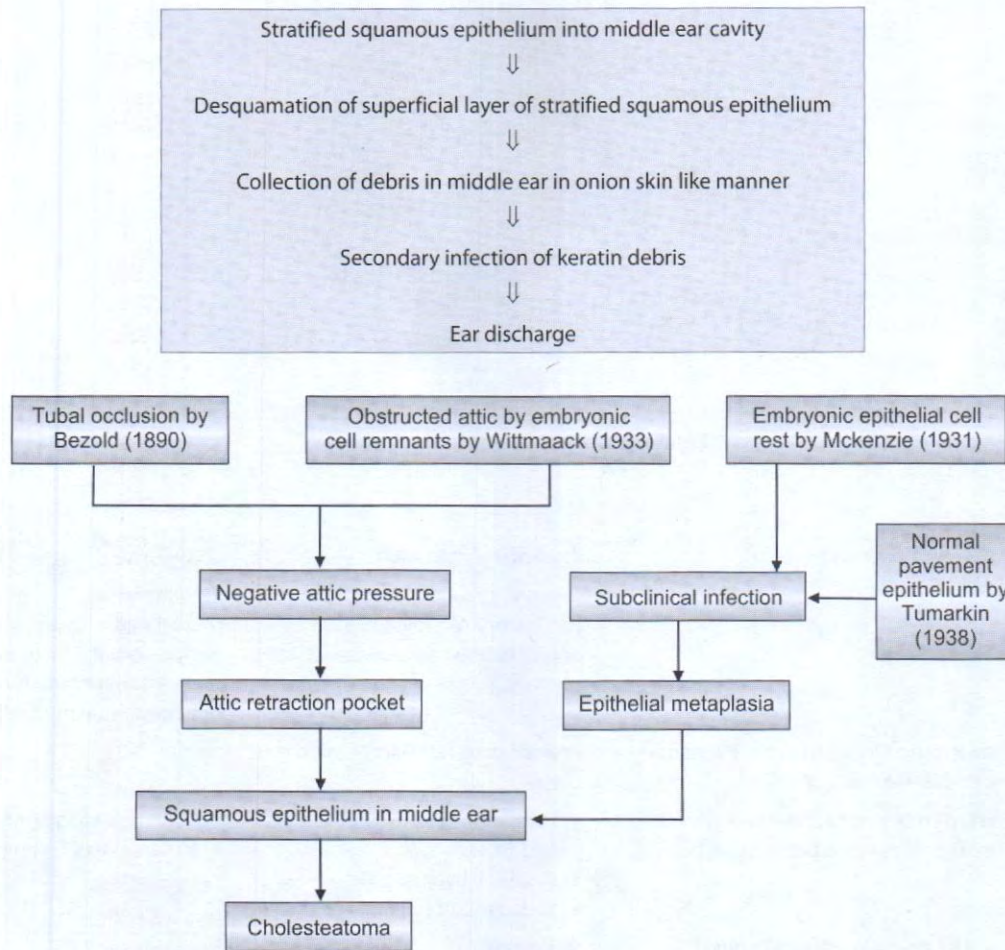


Figure 2: Cholesteatoma—pathogenesis

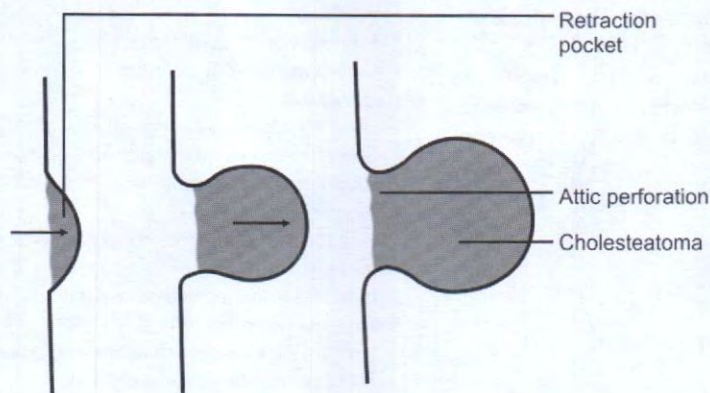


Figure 3: Cholesteatoma—stages of formation

Parts (Morphology)

Matrix	Core
♦ Outer mass of keratinizing squamous epithelium resting on thin stroma of fibrous tissue	♦ Central white mass of keratin debris produced by matrix and arranged like onion skin layers

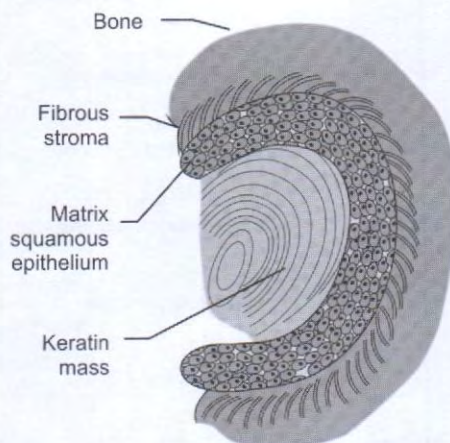


Figure 4: Cholesteatoma—morphology

Features

	<i>Congenital cholesteatoma</i>	<i>Acquired cholesteatoma</i>	
		<i>Primary</i> [No history of previous otitis media or preexisting infection (hence called primary)]	<i>Secondary</i> [Associated with preexisting perforation of pars tensa (especially posterosuperior marginal perforation or large central perforation)]
Location	Anterosuperior quadrant of TM, middle ear, petrous apex and CP angle	Attic or posterior part of middle ear cavity	
Origin	From embryonic epidermal cells and rests in middle ear cleft or temporal bone	<ul style="list-style-type: none"> ♦ Formation of retraction pockets in middle ear ♦ Basal cell hyperplasia ♦ Metaplasia of columnar epithelium 	<ul style="list-style-type: none"> ♦ Migration of squamous epithelium ♦ Metaplasia of columnar epithelium
Clinical features	<ul style="list-style-type: none"> ♦ Usually an incidental finding ♦ Manifests as conductive hearing loss (due to interference with Eustachian tube function or impairment of ossicular chain mobility) ♦ On examination shows white mass behind an intact tympanic membrane ♦ May complicate due to spontaneous rupture through tympanic membrane causing discharging ear 	<p>Symptoms</p> <ol style="list-style-type: none"> Ear discharge <ul style="list-style-type: none"> – Scanty and foul smelling (due to presence of saprophytic infection and osteitis) – Predominantly purulent – Occasionally blood stained Hearing loss <ul style="list-style-type: none"> – Usually progressive conductive deafness – Sometimes sensorineural deafness (due to absorption of toxins through round window membrane) Itching and earache <ul style="list-style-type: none"> – Due to co-existing otitis externa or extradural abscess Tinnitus <ul style="list-style-type: none"> – Indicates imminent sensorineural hearing loss Features of bony erosions <ul style="list-style-type: none"> – Vertigo, if lateral semicircular canal eroded – Facial palsy, if facial canal eroded <p>Signs</p> <ul style="list-style-type: none"> ♦ Foul smelling ear discharge in external canal ♦ Granulation tissue in posterosuperior part of deep meatus ♦ Destruction of outer attic wall ♦ Perforation of tympanic membrane in attic, margin or totally ♦ Cholesteatoma seen as cotton wool through perforation ♦ Mastoid tenderness ♦ Associated sagging of posterior superior meatal wall ♦ Hearing test reveal conductive deafness or sometimes sensorineural deafness 	

Contd...

Contd...

	<i>Congenital cholesteatoma</i>	<i>Acquired cholesteatoma</i>
Diagnostic criteria (Derlacki and Clemis)	<ul style="list-style-type: none"> ♦ No history of previous episodes of middle ear disease ♦ Intact and normal eardrum ♦ Purely an incidental finding ♦ If discharge and eardrum perforation is present then it should be contrued that congenital cholesteatoma has managed to erode TM 	
Staging (Nelson's staging)	<ul style="list-style-type: none"> ♦ Stage I: Involvement of mesotympanum without involvement of incus/stapes ♦ Stage II: Involvement of mesotympanum/ attic with erosion of ossicles without extension into mastoid cavity ♦ Stage III: Involvement of mesotympanum with mastoid extension 	
Lab diagnosis	<ul style="list-style-type: none"> ♦ CT confirms diagnosis 	<ul style="list-style-type: none"> ♦ Culture sensitivity of ear discharge ♦ Rigid otoendoscopy ♦ Audiogram ♦ X-ray mastoid <ul style="list-style-type: none"> – Shows sclerosis with presence of cavity ♦ CT scan <ul style="list-style-type: none"> – Blunting of scutum – Erosion and destruction of lateral attic wall (loss of normal figure of 8) – Widening of aditus – Displacement and destruction of ossicles – Labyrinthine fistula formation – Erosion into facial canal – Erosion of posterior and roof of external auditory canal – Dehiscence of tegmen tympani and sinus plate ♦ MRI <ul style="list-style-type: none"> – Provides additional information regarding soft tissues

June 2013 (RS2 & 3)

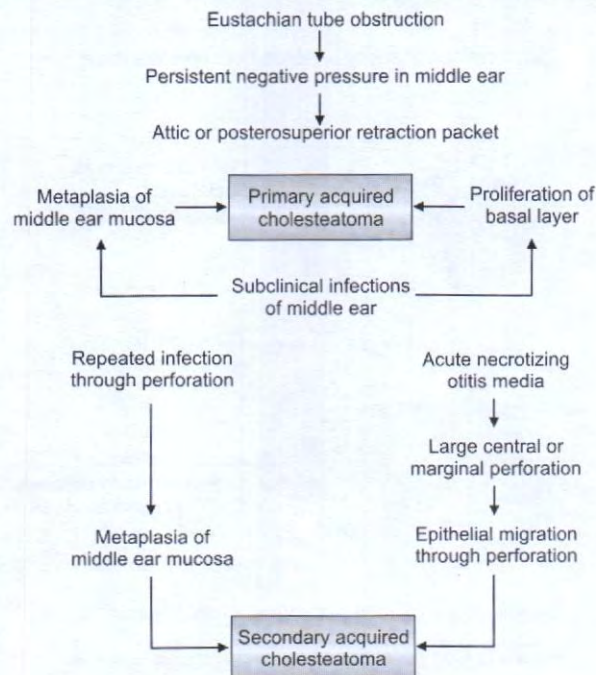


Figure 5: Cholesteatoma—pathogenesis

Treatment

Objectives (Principles)	Conservative	Operative (Main line of treatment)
<ul style="list-style-type: none"> To make ear safe and dry To restore or improve hearing To maintain normal anatomical appearance of ear by avoiding open mastoid cavity 	<p>Indications</p> <ul style="list-style-type: none"> Cholesteatoma with only hearing loss Unfit for surgery <p>Techniques</p> <ol style="list-style-type: none"> Topical antibiotics and steroids 5-fluorouracil 	<p>a. Modified radical mastoidectomy</p> <p>Aims</p> <ul style="list-style-type: none"> To exteriorize disease To create adequate ventilation for middle ear cavity To create permanent skin lined cavity exposed to exterior
Fate	Spread	
<ul style="list-style-type: none"> Cholesteatoma has property to destroy bone causing destruction of ear ossicles, erosion of bony labyrinth, facial nerve canal, sinus plate or tegmen tympani Bony destruction initially occurs along path of least resistance followed by enzymatic destruction due to collagenase, acid phosphatase and proteolytic enzymes liberated by osteoclasts and mononuclear inflammatory cells 	<ul style="list-style-type: none"> Attic cholesteatoma may extend backwards into aditus, antrum and mastoid, downward into mesotympanum, medially surrounding incus and/or head of malleus 	

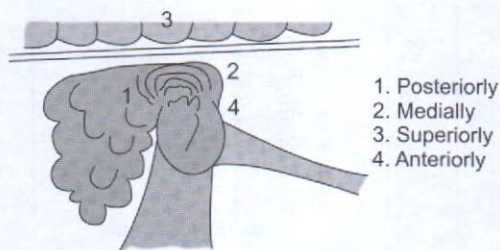
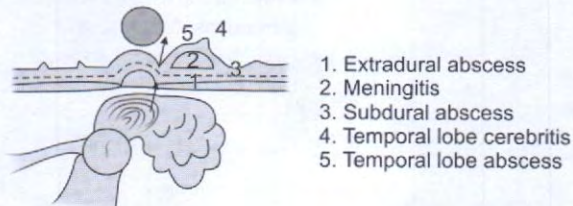
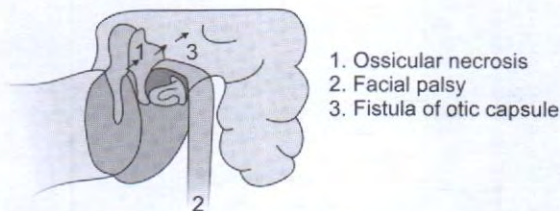


Figure 6: Spread of cholesteatoma

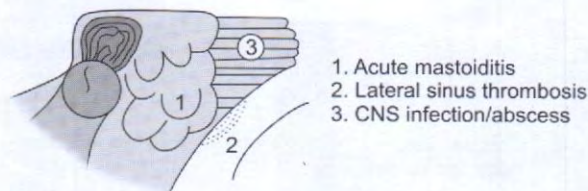
Complications of Spread



Complications of superior spread of cholesteatoma



Complications of medial spread of cholesteatoma



Complications of posterior spread of cholesteatoma

Figure 7: Complications of spread of cholesteatoma

2. Classify the causes of epistaxis. Outline the management of epistaxis in a 60-year-old man.

Refer Question No. 1 June 2009 (RS2).

■ SHORT ESSAYS**3. Olfactory area.**

Refer Question No. 3 June 2012 (RS2).

4. Indications and complications of esophagoscopy.

Refer Question No. 4 June 2012 (RS2).

5. Thyroglossal cyst.

Refer Question No. 5 June 2012 (RS2).

6. Organ of Corti.

Refer Question No. 6 June 2012 (RS2).

7. Gradenigo's syndrome.

Refer Question No. 3 December 2009 (RS2).

8. Dangerous area of the face.

Refer Question No. 8 June 2012 (RS2).

9. Functional endoscopic sinus surgery.

Refer Question No. 12 December 2007 (RS2).

10. Etiology and clinical features of vasomotor rhinitis.

Refer Question No. 10 June 2012 (RS2).

11. Cochlear implant.

Refer Question No. 11 June 2012 (RS2).

12. Cortical mastoidectomy.

Refer Question No. 12 June 2012 (RS2).

■ SHORT ANSWERS**13. Keratosis obturans.**

Refer Question No. 21 June 2008 (RS2).

14. Acoustic trauma.

Refer Question No. 14 June 2012 (RS2).

15. Rhinolith.

Refer Question No. 17 December 2008 (RS2).

16. Osteomeatal complex.

Refer Question No. 9 December 2009 (RS2).

17. Adenoid facies.

Refer Question No. 7 June 2016 (RS2).

18. Globus hystericus.

Refer Question No. 18 June 2012 (RS2).

19. Lingual thyroid.

Refer Question No. 6 June 2008 (RS2).

20. Stridor.

Refer Question No. 1 June 2016 (RS2).

21. Little's area.

Refer Question No. 20 June 2011 (RS2).

22. Bezold's abscess.

Refer Question No. 9 June 2008 (RS2).

MBBS PHASE III EXAMINATION

DECEMBER 2013

(Revised Scheme 2 & 3)

LONG ESSAYS

1. What is cholesteatoma? Describe the clinical features and management of atticoantral type of chronic suppurative otitis media.

- Atticoantral type of chronic suppurative otitis media is long standing chronic infection of posterosuperior part of middle ear cavity (attic, antrum, posterior tympanum and mastoid), characterized by presence of destructive cholesteatoma
- Also called tympanomastoid otitis media
- Also called unsafe or dangerous type because of risk of serious complications caused by bone eroding property of cholesteatoma.

Classification

Inactive (atelectatic ear)	Active
<ul style="list-style-type: none">♦ Retraction pockets present♦ No discharge	<ul style="list-style-type: none">♦ Cholesteatoma present which erodes bone, forms granulation tissue♦ Purulent offensive discharge

Etiopathogenesis

- Atticoantral type of chronic suppurative otitis media is associated with cholesteatoma formation
- Cholesteatoma is a sac lined by keratinizing stratified squamous epithelium containing keratinized desquamated epithelium debris in middle ear or mastoid resting on a fibrous tissue layer, i.e. it is skin in wrong place.

Etiology (Genesis of Cholesteatoma)

a. Congenital theory	<ul style="list-style-type: none">♦ Presence of congenital cell nests, trapped in parietal bone or elsewhere in skull
b. Wittmaack's theory (Retraction pockets)	<ul style="list-style-type: none">♦ Eustachian tube malfunction forms retraction pockets in attic or posterosuperior quadrant of eardrum which lines wall of middle ear with help stratified squamous epithelium on outer surface of eardrum♦ This retraction pockets hamper normal migratory action of epithelium of external auditory meatus resulting in accumulation of desquamated epithelium in attic♦ This causes pressure necrosis of tympanic membrane forming cholesteatoma of middle ear
c. Ruedi's theory (Basal cell hyperplasia)	<ul style="list-style-type: none">♦ Laying down of keratinizing squamous epithelium by basal cells of germinal layer of skin under influence of infection
d. Habermann's theory (Epithelial in growth or theory of migration)	<ul style="list-style-type: none">♦ Invasion of epithelium from external canal or outer surface of tympanic membrane through a preexisting perforation (especially marginal type) with destroyed annulus tympanicus or due to absence of middle fibrous layer of drum in attic
e. Sade's theory (Metaplasia)	<ul style="list-style-type: none">♦ Metaplasia of middle ear mucosa into squamous epithelium due to repeated infections
f. Implantation theory	<ul style="list-style-type: none">♦ Due to implantation of squamous epithelium in middle ear during surgery

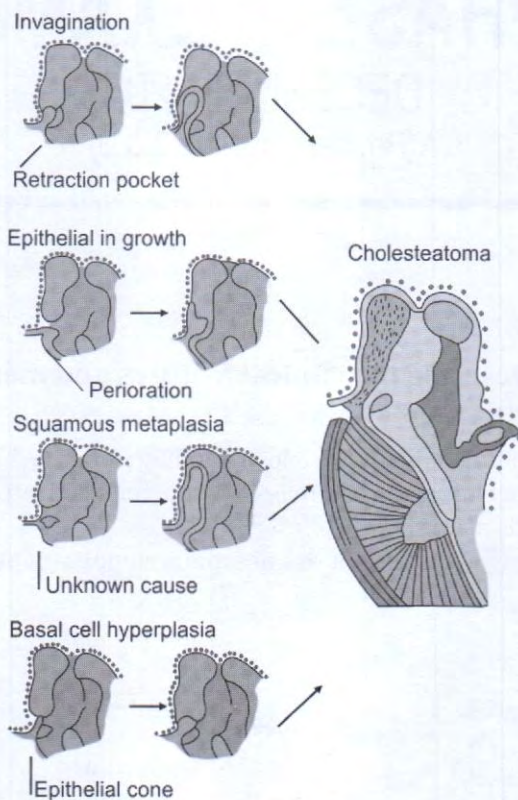
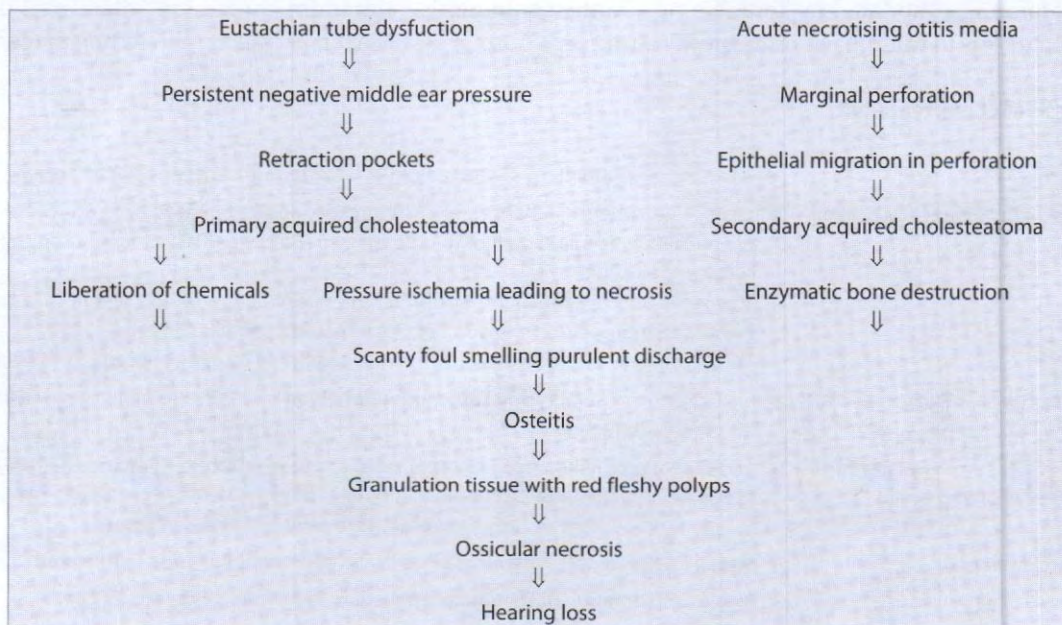


Figure 1: Cholesteatoma—theories of genesis

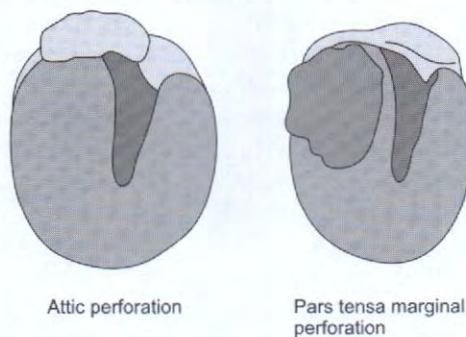
Pathogenesis



Clinical Features

- Insidious onset.

Symptoms	
a. Ear discharge	<ul style="list-style-type: none"> ♦ Foul smelling (like dead mouse/rotten fish) ♦ Purulent and continuous ♦ Yellowish, brownish or greenish ♦ Scanty ♦ May be blood stained due to granulations ♦ Not affected by upper respiratory tract infections
b. Hearing loss	<ul style="list-style-type: none"> ♦ Progressive and mild to moderate (rarely exceeds 50 dB) ♦ Conductive hearing loss initially but later develops sensorineural hearing-loss in long-standing cases due to spread of infection to labyrinth ♦ Sometimes patients hear better in presence of ear discharge (Round window shielding effect) due to maintenance of phase differential by discharge
c. Ear ache	<ul style="list-style-type: none"> ♦ Caused by complications of chronic otitis media like: <ul style="list-style-type: none"> – Acute otitis media supervening on chronic otitis media – Acute otitis externa – Impending dangerous complication like mastoiditis or intracranial complication
d. Swelling behind ear	♦ Mastoid abscess in may cause swelling behind ear with signs of acute inflammation
Signs	
a. TM perforation	<ul style="list-style-type: none"> ♦ Attic perforation or marginal perforation in posterosuperior quadrant of pars tensa ♦ Marginal perforation associated with cholesteatoma
b. Retraction pockets	<ul style="list-style-type: none"> ♦ Evident as invagination of TM in attic or posterosuperior area of pars tensa ♦ Pocket is shallow and self cleansing in early stages but become deep with accumulated keratin mass later thus getting infected
c. Cholesteatoma	♦ Whitish cholesteatoma flakes seen in retraction pockets through perforation
d. Osteitis	♦ Involves outer attic wall and posterosuperior margin of tympanic ring and surrounded by granulation tissue
e. Granulation tissue	<ul style="list-style-type: none"> ♦ Seen in posterosuperior part of deep meatus ♦ Basically is low grade osteitis of mastoid bone ♦ Surrounds area of osteitis and fill up attic, antrum, posterior tympanum and mastoid
f. Aural polyp	<ul style="list-style-type: none"> ♦ Smooth, pink and fleshy polyp due to prolapse of edematous and inflamed mucosa ♦ Protrudes through perforation into external canal
g. Ossicular necrosis	<ul style="list-style-type: none"> ♦ Common ♦ May be limited to long process of incus or may also involve superstructure of stapes, handle of malleus or entire ossicular chain ♦ Sometimes, cholesteatoma may bridge gap caused by destroyed ossicles thus hearing loss is not apparent (cholesteatoma hearer)
h. Cholesterol granuloma	<ul style="list-style-type: none"> ♦ Mass granulation tissue with foreign body giant cells surrounding cholesterol crystals ♦ Commonly located in mesotympanum ♦ Appears as dark brown gelatinous material, often with bone destruction ♦ Appears dark blue or black behind intact tympanic membrane

**Figure 2:** Tympanic membrane perforations

Investigations

Examination under microscope	Swab of ear discharge	Radiology	Hearing tests (Assess type and degree of hearing loss)
<ul style="list-style-type: none"> To assess presence of granulations, in-growth of squamous epithelium from edges of perforation, status of ossicular chain, tympanosclerosis and adhesions 	<ul style="list-style-type: none"> For culture and sensitivity 	<ul style="list-style-type: none"> Mastoid X-ray (lateral view) shows sclerosis with area of bone destruction, i.e. cavity formation Temporal bone CT scan to assess extent of destruction by cholesteatoma (investigation of choice) MRI provides additional information 	<ul style="list-style-type: none"> i. Tuning fork tests <ul style="list-style-type: none"> Rinne's negative Weber lateralized to affected side Absolute conduction test normal ii. Pure tone audiometry <ul style="list-style-type: none"> Mild to moderate hearing loss (<60 dB)

Treatment

Objectives

- To make ear safe and dry (primary aim)
- To restore or improve hearing
- To maintain normal anatomical appearance of ear by avoiding open mastoid cavity.

Conservative

- Limited role.

Indications	Techniques
<ul style="list-style-type: none"> Cholesteatoma in only hearing ear Small cholesteatoma Unfit for surgery 	<ul style="list-style-type: none"> i. 5-fluorouracil—topically ii. Chemical cautery using silver nitrate or trichloroacetic acid

Operative

- Mainstay of treatment.

	Canal wall down procedures			Canal wall up procedures
Principle	Leave mastoid cavity open into external auditory canal so that diseased area is fully exteriorized			Removal of disease by combined approach through meatus and mastoid but retaining posterior bony meatal wall intact, thereby avoiding open mastoid cavity
Techniques	Atticotomy	Radical mastoidectomy	Modified radical mastoidectomy (described below)	Combined approach tympanoplasty (mastoidectomy + tympanoplasty)
Principle	<ul style="list-style-type: none"> Consists of removal of triangular area of bone bounded superiorly along suprameatal crest, inferiorly along posterosuperior canal wall with apex pointing towards zygoma followed by removal diseased tissue 	<ul style="list-style-type: none"> Consists of removal of posterior meatal wall thus converting entire area of middle ear, attic, antrum and mastoid into a single cavity by removing all remnants of tympanic membrane, ossicles (except footplate of stapes) and mucoperiosteal lining 	<ul style="list-style-type: none"> Consists of removal of disease process localized to attic and antrum followed by exteriorization of entire area by removal of posterior meatal and lateral attic walls 	<ul style="list-style-type: none"> Consists of modified mastoidectomy followed by reconstructive procedures for restore hearing with or without tympanic membrane grafting

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	Canal wall down procedures			Canal wall up procedures
Advantages	<ul style="list-style-type: none"> Intermediate risk of otorrhea 	<ul style="list-style-type: none"> Low chance of recurrent pars flaccida and pars tensa cholesteatoma 	<ul style="list-style-type: none"> Residual cholesteatoma is visible on follow up Low chance of recurrent pars flaccida and pars tensa cholesteatoma Complete exteriorization of facial recess Second look surgery rarely required 	<ul style="list-style-type: none"> Gives dry ear (low risk of otorrhea) Permits easy reconstruction of hearing mechanism
Disadvantages	<ul style="list-style-type: none"> Risk of recurrent pars flaccida cholesteatoma Second look surgery required 	<ul style="list-style-type: none"> Conductive hearing loss Significant risk of otorrhea 	<ul style="list-style-type: none"> Formation of large mastoid cavity Shallow middle ear Significant risk of otorrhea Alteration of position of pinna Postoperative vertigo due to caloric stimulation of lateral semicircular canal 	<ul style="list-style-type: none"> High incidence of residual or recurrent cholesteatoma Secondary look surgery required



A. Enlarging external bony canal



B. Bony covering retraction pocket removed



C. Exposing retraction pocket

Figure 3: Limited atticotomy

Modified Radical Mastoidectomy (Bondy's Operation)

- Surgery of choice.

Principle

- Modification of radical mastoidectomy to preserve as much as hearing mechanism
- Consists of removal of disease process localized to attic and antrum followed by exteriorization of entire area by removal of posterior meatal and lateral attic walls.

Indications	Contraindications
<ul style="list-style-type: none"> Cholesteatoma confined to attic and antrum Localized chronic otitis media 	<ul style="list-style-type: none"> Relative contraindications like diabetes, hypertension, bleeding disorders

Anesthesia

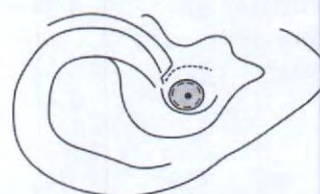
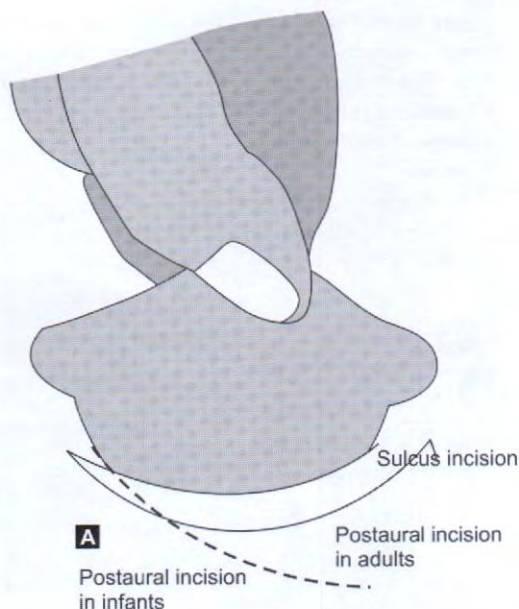
- General anesthesia.

Position

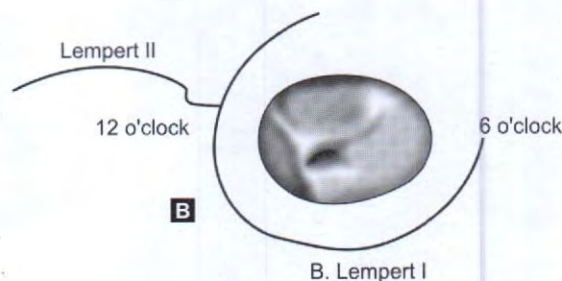
- Supine with face turned to one sided such that ear to be operated is upper most.

Incision

Postaural (Wilde's)	Endaural (Lempert's)—consists of two incisions	
	♦ Not recommended for extensive disease	
♦ Curved postaural incision about 1 cm behind but parallel to retroauricular sulcus starting at highest attachment of pinna to mastoid tip	<i>Lempert I</i>	<i>Lempert II</i>
	♦ Semicircular incision from 12 o'clock to 6 o'clock in posterior meatal wall at bony cartilagenous junction	♦ Starts from 12 o'clock of Lempert I incision and then passing upwards in curvilinear fashion between tragus and crus of helix



A. Lempert's endaural incision



B. Lempert I

Figures 4A and B: (A) Postaural incision; (B) Lempert's incision

Procedure

- Postincision, elevate soft tissue from bone by a periosteum elevator
- Achieve hemostasis and avoid cutting temporalis muscle
- Expose mastoid by applying self-retaining mastoid retractor
- Identify suprameatal triangle to remove bone of mastoid cortex using hammer and drill or burr thus exposing attic and antrum
- Remove diseased tissue along with cholesteatoma, granulation tissue and unhealthy mucosa
- Preserve incus and head of malleus but remove them if required (when cholesteatoma engulfs them or extends medial to them)
- Remove lateral wall of attic to fully exteriorise attic
- Lower facial ridge as much as possible within safety of facial nerve
- Smoothen mastoid cavity with polishing burr, removing any overhangs followed by irrigation using normal saline
- Remove irreversible damaged tissue from middle ear
- Reconstruct tympanic membrane or ossicular chain if damaged
- Pack cavity with ribbon gauze, impregnated with antibiotics
- Close incision in 2 layers with interrupted sutures
- Apply pressure dressing using mastoid bandage.

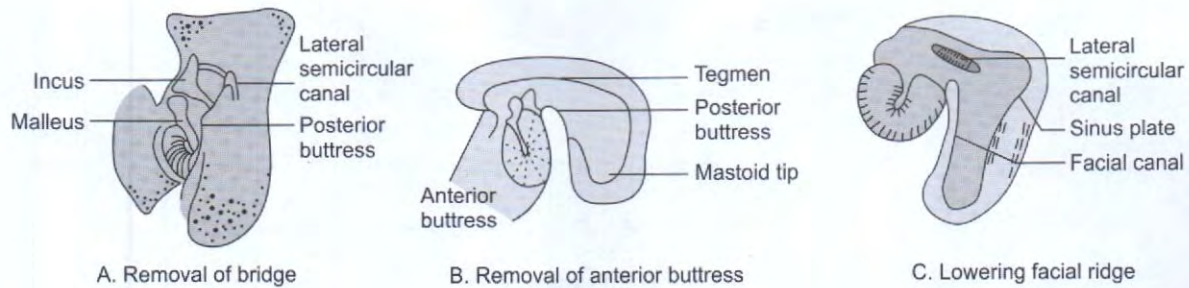


Figure 5: Modified radical mastoidectomy

Postoperative care	Complications
<ul style="list-style-type: none"> Antibiotics for atleast 1wk First dressing on 3–4th day followed by second dressing on 6–7th day Remove stitches after 6 days Regular follow up (every 4–6 months) in first year and then annually for removal any debris or infection 	<ul style="list-style-type: none"> Formation of mastoid cavity which may not heal and continue to discharge (25%) Facial paralysis Perichondritis of pinna Injury to sigmoid sinus with profuse bleeding Injury to dura mater Labyrinthitis, if stapes gets dislocated Postoperative wound infection and wound breakdown
Advantages	Disadvantages
<ul style="list-style-type: none"> Residual cholesteatoma is visible on follow up Low chance of recurrent pars flaccida and pars tensa cholesteatoma Complete exteriorization of facial recess Second look surgery rarely required 	<ul style="list-style-type: none"> Formation of large mastoid cavity Shallow middle ear Significant risk of otorrhea Alteration of position of pinna Postoperative vertigo due to caloric stimulation of lateral semicircular canal

2. Describe the etiology, clinical features and management of nasal polyposis.

- Nasal poly is an idiopathic inflammatory condition presenting as prolapsed, non-neoplastic pedunculated mass of edematous mucosa of nose or nasal sinuses.

Types

- Common types
 - Ethmoidal polyps
 - Antrochoanal polyps.
- Rare types
 - Sphenchoanal
 - True choanal.

A. Ethmoidal

- Ethmoidal polyps are non-neoplastic masses of hypertrophied edematous mucosa of ethmoid sinuses.

Etiopathogenesis

Bernoulli's phenomenon	Polysaccharide change	Vasomotor imbalance	Allergy	Chronic infection
↓	↓	↓	↓	↓
Passage of air through narrow nasal cavity produces negative pressure next to constriction in vicinity of paranasal sinuses	Change in polysaccharide content of ground substance of nasal mucosa	Vasodilatation and increased permeability of vessels		Perilymphangitis and periphlebitis

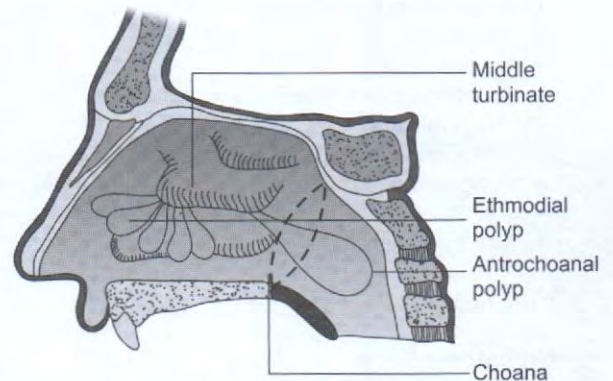


Figure 6: Nasal polyps—types

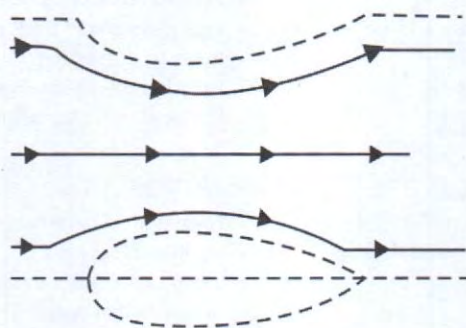
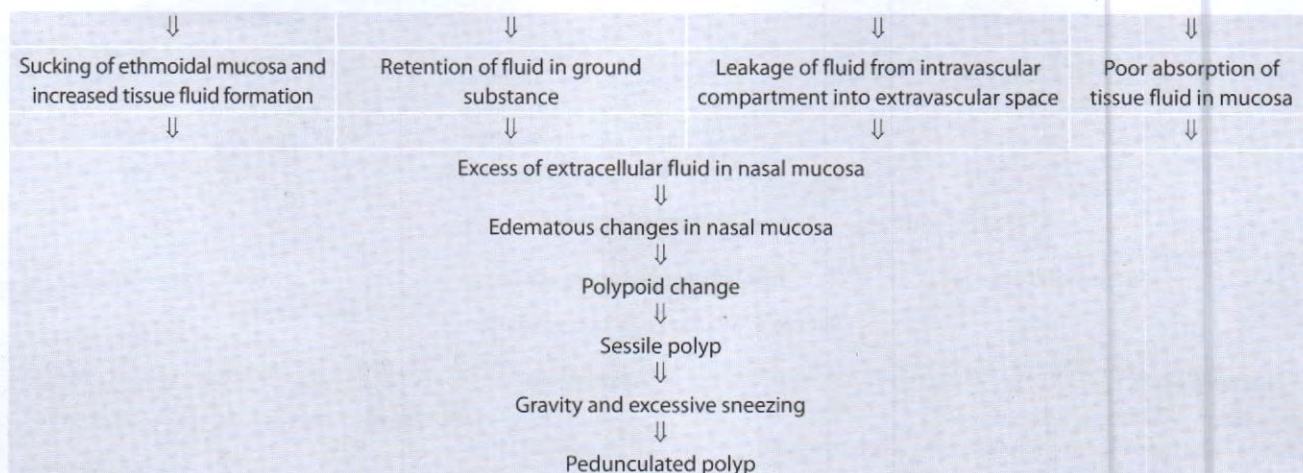


Figure 7: Bernoulli's phenomenon

Predisposing factors	Site of origin (lateral wall of nose)
<ul style="list-style-type: none"> ♦ Chronic rhinosinusitis (both allergic and nonallergic) ♦ Asthma (7%) ♦ Aspirin intolerance (36%) ♦ Cystic fibrosis (20%) due to abnormal mucus ♦ Allergic fungal sinusitis ♦ Kartagener's syndrome (bronchiectasis sinusitis, situs inversus and ciliary dyskinesia) ♦ Young syndrome (sinopulmonary disease and azoospermia) ♦ Churg-Strauss syndrome (asthma, fever, eosinophilia, vasculitis and granuloma) ♦ Nasal mastocytosis 	<ul style="list-style-type: none"> ♦ Uncinate process ♦ Bulla ethmoidalis ♦ Ostia of sinuses ♦ Medial surface and edge of middle turbinate

Pathology

Gross appearance

- Smooth, soft, glistening, pale, grape like mass with ulceration of protruded part.

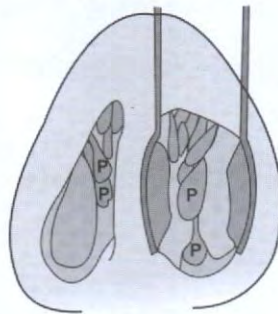
Microscopic examination

- Mucosal covering of ciliated columnar epithelium initially but undergoes metaplastic change to transitional and squamous type on exposure to atmospheric irritation
- Edema of lamina propria
- Increase in intraepithelial glandular structures
- Submucosa contains loose fibrillar stroma and large intercellular spaces filled with serous fluid and scanty blood vessels and nerves
- Submucosal infiltration with IgA, IgG, eosinophils and round cells.

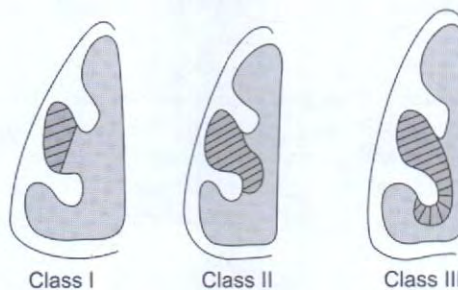
Clinical Features

- Occurs at any age but mostly adults
- Bilateral involvement.

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Nasal stuffiness leading to total nasal obstruction ♦ Loss of smell sensation (total or partial) ♦ Headache (due to associated headache) ♦ Sneezing and watery nasal discharge (due to associated allergy) ♦ Mass protruding from nose ♦ Snoring and mouth breathing 	<ul style="list-style-type: none"> ♦ Smooth, soft, glistening, pale, pearly white or bluish and pulpy grape like masses ♦ May be single or multiple ♦ May be sessile or pedunculated (probe can be passed all around) ♦ Insensitive and do not bleed on touch ♦ Postnasal drip ♦ Cold spatula test reveals reduced or absence of fogging ♦ Purulent discharge in nasal cavity (due to associated sinusitis) ♦ Broadening of nose and increased intercanthal distance (long-standing case)—frog face deformity ♦ Pink, vascular mass with ulceration protruding from nose (simulating neoplasm)

**Figure 8:** Ethmoidal polyposis—anterior rhinoscopy**Staging**

Class I	Class II	Class III
♦ Smaller than middle turbinate	♦ Larger than middle turbinate but smaller than inferior turbinate	♦ Larger than inferior turbinate

**Figure 9:** Nasal polyps—grading**Investigations**

Nasal discharge	Radiography	Diagnostic nasal endoscopy	Biopsy
<ul style="list-style-type: none"> ♦ Show eosinophils in patients with allergy 	<ul style="list-style-type: none"> ♦ X-ray of ethmoidal sinuses and nose show opacity ♦ CT scan of paranasal sinuses to exclude bony erosion and expansion (rules out malignancy) 	<ul style="list-style-type: none"> ♦ To confirm diagnosis and find associated pathology 	<ul style="list-style-type: none"> ♦ To confirm diagnosis and rule of malignancy

Differential Diagnosis

<i>Hypertrophied turbinate</i>	<i>Rhinosporidiosis</i>	<i>Angiofibroma of septum</i>	<i>Transitional cell/squamous papilloma</i>	<i>Meningocele</i>	<i>Malignancy of nose</i>
<ul style="list-style-type: none"> ♦ Pink, tender, firm ♦ Sensitive to touch ♦ No pedicle ♦ Shrinks with decongestants 	<ul style="list-style-type: none"> ♦ Bleeding polypoid mulberry like mass (strawberry like appearance) ♦ Arises from nasal septum ♦ Shows sporangia ♦ Common in people from coastal India 	<ul style="list-style-type: none"> ♦ Single, smooth, red, firm, bleeding polypoid mass ♦ History of recurrent epistaxis ♦ Arises from nasal septum 	<ul style="list-style-type: none"> ♦ Single, friable, pink ♦ Opaque and fleshy ♦ Bleeds on touch 	<ul style="list-style-type: none"> ♦ Soft, cystic polyp like swelling ♦ Common in young children 	<ul style="list-style-type: none"> ♦ Friable mass, bleeds on touch

Treatment

Conservative

<i>Antihistaminics (local and systemic)</i>	<i>Corticosteroids (local and systemic)</i>	<i>Antibiotics</i>
<ul style="list-style-type: none"> ♦ For early polypoid change with edematous mucosa due to allergy ♦ After surgery to prevent recurrence 	<ul style="list-style-type: none"> ♦ In cases resistant to antihistaminics or with asthma ♦ After surgery to prevent recurrence 	<ul style="list-style-type: none"> ♦ In presence of infections along with antihistaminics/steroids

Operative

a. Polypectomy

- Avulsion of polyp using nasal snares for 1-2 pedunculated polyps or Luc's forceps for multiple sessile polyps under local or general anesthesia.

b. Ethmoidectomy

Techniques

	<i>Intranasal</i>	<i>Extranasal (Howarth's)</i>	<i>Transantral (Horgan's)</i>
Indication	♦ Multiple sessile polyps	♦ Recurring polyp after intranasal procedure and ill-defined surgical landmarks	♦ Infection and polypoid changes in maxillary antrum
Procedure	♦ Involves uncapping of ethmoidal air cells through middle meatus route	♦ Involves approaching ethmoidal air cells through medial wall of orbit by an external curved incision medial to medial canthus	♦ Involves approaching ethmoidal air cells through medial wall of antrum and antrum via Caldwell Luc approach
Remarks	<i>Disadvantages</i> <ul style="list-style-type: none"> ♦ Anterior most ethmoidal air cells (aggr nasi) cannot be removed completely ♦ Can damage orbital contents, optic nerve or anterior cranial fossa 	<i>Advantages</i> <ul style="list-style-type: none"> ♦ Complete removal of diseased air cells ♦ Less chances of damage to surrounding structures <i>Disadvantages</i> <ul style="list-style-type: none"> ♦ External scar 	



Figure 10: Ethmoidectomy

- c. Functional endoscopic sinus surgery (FESS)—treatment of choice
- Involves removal of polyps and ethmoidal air cells and provision of drainage and ventilation to other involved sinuses using endoscopes under general anesthesia.

Advantages

- Helps preserve normal function of sinuses.

B. Antrochoanal

- Antrochoanal polyp non-neoplastic mass of hypertrophied edematous mucosa of maxillary antrum near its accessory ostia
- Also called Killian's polyp.

Etiopathogenesis

Etiology	Predisposing factors	Pathogenesis
<ul style="list-style-type: none"> ♦ Idiopathic ♦ Faulty development of maxillary ostium (Proetz) ♦ Bernoulli's phenomenon 	<ul style="list-style-type: none"> ♦ Nasal allergy ♦ Sinusitis 	<p>Prolapsed mucosa hanging down from roof of antrum</p> <p>↓</p> <p>Edema of mucosa</p> <p>↓</p> <p>Sagging down of mucosa forming neck at its base</p> <p>↓</p> <p>Formation of polyp</p> <p>↓</p> <p>Gradual filling of antrum</p> <p>↓</p> <p>Overflowing of polyp through ostia to hiatus semilunaris</p> <p>↓</p> <p>Grows downward and backward following direction of ostia to reach choana (Dumb-bell shaped nasal polyp with constriction at maxillary antrum)</p> <p>↓</p> <p>Enters nasopharynx through choana</p> <p>↓</p> <p>Hangs down into oropharynx and laryngopharynx (Trifoliate shaped nasal polyp)</p>

Pathology

Gross appearance (parts)	Microscopic examination
<ul style="list-style-type: none"> ♦ Large, smooth, soft, globular, grayish mass consisting of three parts <ul style="list-style-type: none"> - Antral Thin stalk - Choanal Round and globular - Nasal Flat from side to side 	<ul style="list-style-type: none"> ♦ Lined by respiratory epithelium with normal basement membrane ♦ ↑ in intraepithelial glandular structures ♦ Submucosa contains loose fibrillar stroma and large intercellular spaces filled with serous fluid and scanty blood vessels and nerves

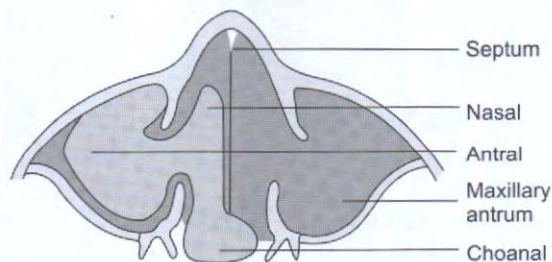


Figure 11: Choanal polyp—parts

Clinical Features

- Common in children and young adults
- Usually unilateral.

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Nasal obstruction usually during expiration – initially unilateral but becomes bilateral once polyp grows into nasopharynx and obstructs opposite choana ♦ Thick and dull voice (due to hyponasality) ♦ Muroid nasal discharge on one or both sides ♦ Headache or heaviness in head on affected side ♦ Stridor (if obstructs laryngopharynx) 	<ul style="list-style-type: none"> ♦ Single, large, smooth, soft, globular, translucent, grayish mass filling choana and nasopharynx ♦ Covered with nasal discharge ♦ Can be moved up and down with probe but probe cannot be passed around it ♦ Postnasal drip ♦ Pink congested look on exposed part if protrudes from nostrils ♦ Pear-shaped swelling hanging behind soft palate if extended into oropharynx

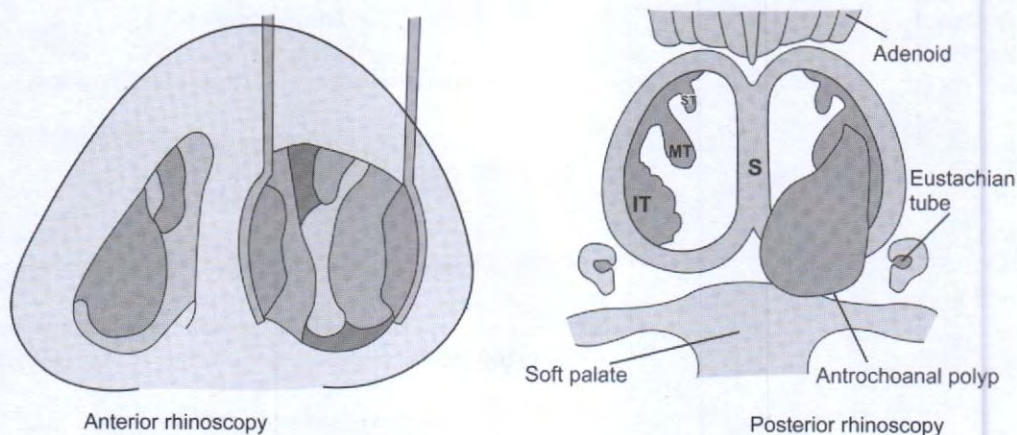


Figure 12: Antrochoanal polyp

Staging

Grade I	Grade II	Grade III
♦ Confined to antrum	♦ Polyp in nasal cavity	♦ Polyp extending to nasopharynx

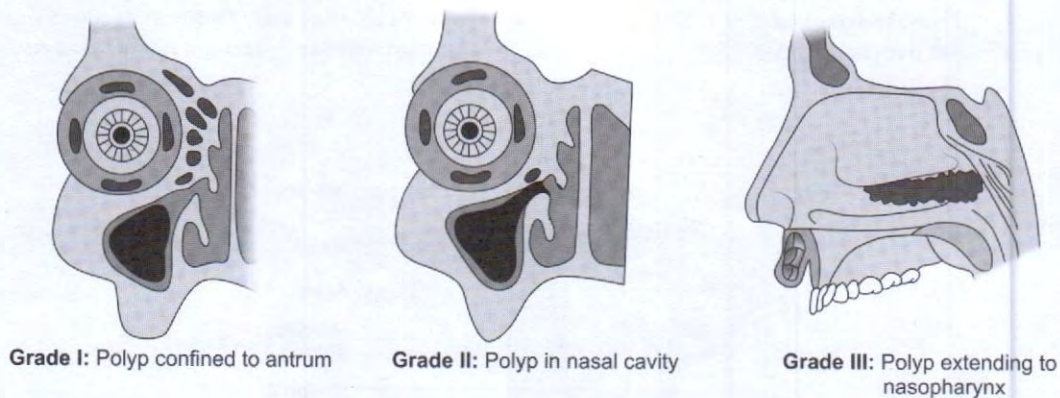


Figure 13: Staging of antrochoanal polyp

Investigations

Radiology	Diagnostic nasal endoscopy	Biopsy
<ul style="list-style-type: none"> ♦ X-ray of paranasal sinuses and nose show opacity of involved antrum and nose ♦ X-ray (lateral view) of neck reveals globular swelling in postnasal space with column of air behind and above it (crescent sign) ♦ CT scan of osteomeatal complex to identify attachment site 	<ul style="list-style-type: none"> ♦ To confirm diagnosis and find associated pathology 	<ul style="list-style-type: none"> ♦ To confirm diagnosis and rule of malignancy

Differential Diagnosis

Hypertrophied posterior end of turbinate	Nasopharyngeal rhinosporidiosis	Nasopharyngeal angiofibroma	Transitional cell/squamous papilloma	Nasopharyngeal malignancy	Hamartoma
<ul style="list-style-type: none"> ♦ Pink, tender, firm ♦ Sensitive to touch ♦ No pedicle ♦ Shrinks with decongestants 	<ul style="list-style-type: none"> ♦ Bleeding polypoid mulberry-like mass (strawberry-like appearance) ♦ Arises from nasal septum ♦ Shows sporangia ♦ Common in people from coastal India 	<ul style="list-style-type: none"> ♦ Occurs only in young males ♦ History of recurrent epistaxis ♦ Single, smooth, red, firm, bleeding polypoid mass 	<ul style="list-style-type: none"> ♦ Single, friable, pink ♦ Opaque and fleshy ♦ Bleeds on touch 	<ul style="list-style-type: none"> ♦ Affects over 40 years ♦ Irregular, friable mass, bleeds on touch ♦ Lymph node metastasis 	<ul style="list-style-type: none"> ♦ Developmental malformation ♦ Benign, tumor like growth of tissue

Treatment

Conservative	Operative		
	Polypectomy	Caldwell Luc operation	Functional endoscopic sinus surgery (FESS)—treatment of choice
<ul style="list-style-type: none"> ♦ Antibiotics <ul style="list-style-type: none"> – To control associated sinusitis 	<ul style="list-style-type: none"> ♦ Avulsion of polyp using cold wire snares through nasal or oral route under local or general anesthesia ♦ Loop of snare wire is taken to base of polyp and shortened ♦ Polyp is avulsed from base by jerking it 	<p><i>Indications</i></p> <ul style="list-style-type: none"> ♦ Recurrent polyp ♦ Co-existent maxillary sinusitis <p><i>Contraindications</i></p> <ul style="list-style-type: none"> ♦ Children below 14 years (upper 2nd molar needs to be erupted) 	<ul style="list-style-type: none"> ♦ Involves removal of polyps and provision of drainage and ventilation to other involved sinuses using endoscopes under GA <p><i>Advantages</i></p> <ul style="list-style-type: none"> ♦ Helps preserve normal function of sinuses

■ SHORT ESSAYS

3. Rhinosporidiosis.

Refer Question No. 7 June 2010 (RS2).

4. Clinical features and management of rhinoscleroma.

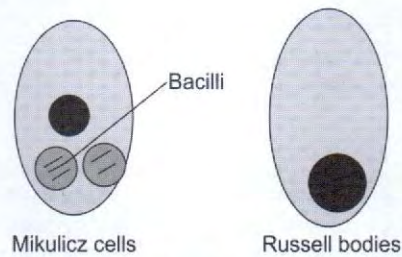
- Rhinoscleroma is chronic progressive granulomatous infection of nose characterized by sclerosis and stenosis of nose.

Etiology

Causative agent	Predisposing factors
<ul style="list-style-type: none"> ♦ <i>Klebsiella rhinoscleromatis</i> (Frisch bacillus) 	<ul style="list-style-type: none"> ♦ Age >20 years ♦ Geography <ul style="list-style-type: none"> – Seen in rural areas of Central Europe and South America – In India, Madhya Pradesh, Mumbai, Delhi, Uttar Pradesh and Punjab ♦ Low socioeconomic status

Pathology

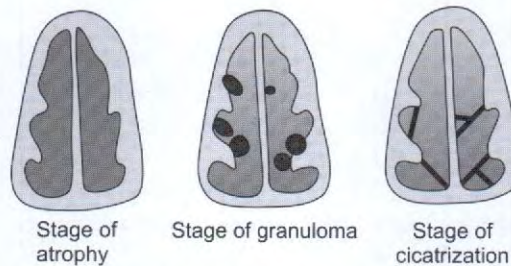
- Infiltration of nasal submucosa by inflammatory cells and pathognomonic Mikulicz cells and Russell bodies
- Mikulicz cells are large foam cells with a central nucleus and vacuolated cytoplasm containing acid fast Frisch bacilli.

**Figure 14:** Thinoscleroma—pathology

- Russell bodies are cells resembling plasma cells with eccentric nucleus and deep staining cytoplasm.

Stages

Atrophic stage	Granulomatous (nodular) stage	Cicatrical (fibrotic) stage
♦ Resembles atrophic rhinitis	♦ Formation of painless, nonulcerative granulomatous nodules	♦ Extension of disease into nasopharynx, oropharynx, subglottic region of larynx, trachea and bronchi

**Figure 15:** Rhinoscleroma—stages**Clinical Features**

	Symptoms	Signs
Early stage	♦ Foul smelling purulent nasal discharge ♦ Crusting of nasal discharge	♦ Painless, nonulcerative, bluish red nodules of rubbery consistency at mucocutaneous junction of septum ♦ Firm and woody feel to lower part of nose and upper lip (due to subdermal infiltration)
Later stage	♦ Progressive blocking of nose on both sides ♦ Distortion of upper lip ♦ Respiratory distress (due to subglottic stenosis)	♦ Adhesions of nose, nasopharynx, oropharynx ♦ Tapir's nose (characteristic change in shape and contour of nose) ♦ Subglottic stenosis

Investigations

Levim test	Biopsy	Culture
♦ Complement fixation test against <i>K. rhinoscleromatis</i> bacilli	♦ Reveals nasal submucosa infiltrated with plasma cells, lymphocytes, eosinophils, Mikulicz cells and Russell bodies	♦ Culture of causative organism from biopsy material

Differential Diagnosis

Tertiary syphilis	Atrophic rhinitis
	♦ Resembles early atrophic stage ♦ Characterized by pale nasal mucosa

Treatment

Conservative	Operative
a. Antibiotics – 1 g streptomycin along with 2 g tetracycline per day for 4–6 weeks b. Steroids – To reduce fibrosis	♦ To establish airway and correct nasal deformity

5. Treatment of secretory otitis media.

- Secretory otitis media (OME) is insidious condition characterized by accumulation of non-purulent sterile effusion in middle ear cavity
- Also called serous otitis media, nonsuppurative otitis media, otitis media with effusion, mucoid otitis media, glue ear.

Etiopathogenesis

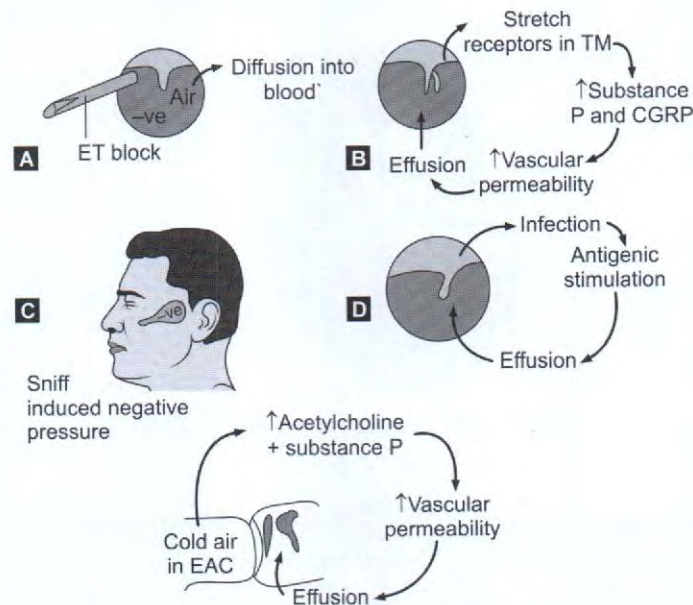


Figure 16: Pathogenesis of OME

	Malfunctioning of Eustachian tube (Results in failure of aeration of middle ear and inability to drain fluid)	Increased secretory activity of middle ear mucosa
Causes	<ul style="list-style-type: none"> ♦ Adenoid hyperplasia ♦ Chronic rhinitis and sinusitis ♦ Chronic tonsillitis (mechanical obstruction to movement of soft palate due to enlarged tonsils thus interfering with physiological opening of Eustachian tube) ♦ Benign and malignant tumors of nasopharynx ♦ Palatal defects like cleft palate, palatal paralysis 	<ul style="list-style-type: none"> ♦ Allergy to inhalants or foodstuffs results in increased secretory activity of middle ear mucosa (which acts as shock organ) and also results in blockage of Eustachian tube by edema ♦ Unresolved otitis media due to inadequate treatment results in low grade infection which may act as stimulant for mucosa to secrete more fluid ♦ Adeno- and rhinoviral infections of upper respiratory tract may invade middle ear stimulating its mucosa to increase secretory activity

Pathology

- Increase in number of goblet cells and mucous glands in middle ear mucosa
- Edema of Eustachian tube.

Clinical Features

- Seen commonly in school-going children of age group 5–8 years.

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Hearing loss <ul style="list-style-type: none"> – Presenting and sometimes only symptom – Insidious in onset – Conductive deafness – Rarely exceeds 40 dB – Vary in degree with changing positions of head ♦ Mild earaches ♦ Delayed and defective speech development (due to hearing loss) ♦ Sensation of fluid moving in ear ♦ Tinnitus may be present 	<ul style="list-style-type: none"> ♦ Tympanic membrane <ul style="list-style-type: none"> – Dull and opaque – Yellow, grey or bluish in color – Loss of light reflex – Show varying degree of retraction but sometimes fully or slightly bulged in posterior part due to effusion – Appearance of thin leash of blood vessel along handle of malleus or at periphery of tympanic membrane – Restricted mobility ♦ Exudate <ul style="list-style-type: none"> – Often thick and viscid but sometimes thin and serous – Fluid level and air bubbles (on Valsalva maneuver) when fluid is thin and tympanic membrane transparent

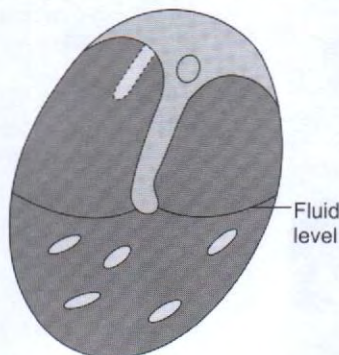


Figure 17: Fluid level and air bubbles in OME

Investigations

Tuning fork tests	Audiometry	Impedance audiometry	X-ray mastoid
<ul style="list-style-type: none"> ♦ Reveals conductive hearing loss 	<ul style="list-style-type: none"> ♦ Demonstrates conductive hearing loss of 20–40 dB ♦ Sometimes associated with sensorineural hearing loss (due to fluid pressing on round window membrane) 	<ul style="list-style-type: none"> ♦ Reduced compliance and flat curve (B curve) with shift to negative side (indicates presence of fluid) 	<ul style="list-style-type: none"> ♦ Clouding of air cells due to fluid

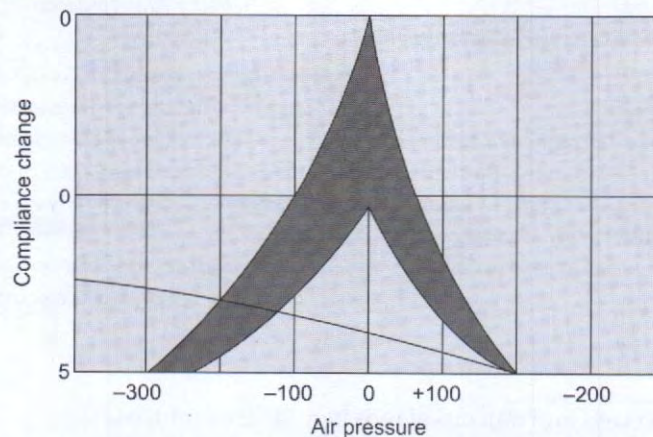
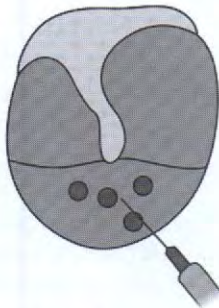


Figure 18: Flat curve with shift in compliance to negative side

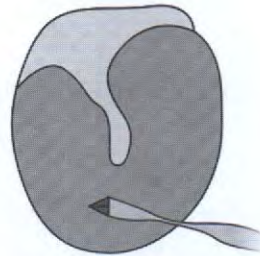
Treatment

Conservative

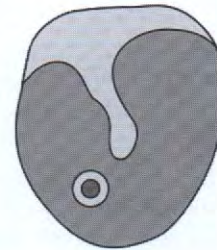
Decongestants	Antiallergic measures	Antibiotics	Middle ear aeration
<ul style="list-style-type: none"> To relieve edema of Eustachian tube <i>Drugs and dosage</i> <ul style="list-style-type: none"> 1% ephedrine nasal drops (0.5% children) 30 mg pseudoephedrine BD 	<ul style="list-style-type: none"> Antihistaminics or steroids in case of allergy Desensitization 	<ul style="list-style-type: none"> Used in case of upper respiratory tract infections or unresolved acute supportive otitis media 	<ul style="list-style-type: none"> Repeated Valsalva maneuver especially adults Chewing gum to encourage repeated swallowing in children to open tube Politzerization or Eustachian tube catheterisation



Simple aspiration



Myringotomy incision

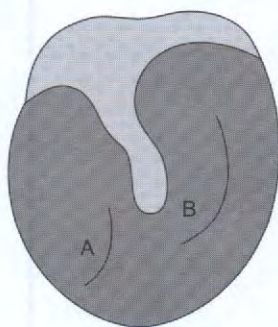


TT in position

Figure 19: Treatment of OME

Operative

	Indications	Procedure
a. Myringotomy	<ul style="list-style-type: none"> Thick and viscid fluid Failure of conservative treatment 	<ul style="list-style-type: none"> Make a radial incision in anteroinferior quadrant of tympanic membrane (sometimes addition incision in anterosuperior quadrant to aspirate glue like secretions) Instill saline or chymotrypsin solution (mucolytic agent) to liquefy thick mucus Aspirate fluid in middle ear cavity with suction
b. Grommet insertion	<ul style="list-style-type: none"> Recurrent serous otitis media even after myringotomy 	<ul style="list-style-type: none"> Insert a grommet through a radial incision in anteroinferior quadrant of TM Retain grommet in place for weeks or months
c. Tympanotomy or cortical mastoidectomy	<ul style="list-style-type: none"> To remove loculated thick fluid or other associated pathologies like cholesterol granuloma 	
d. Surgical treatment of underlying cause		<ul style="list-style-type: none"> Adenoidectomy, tonsillectomy, antral wash, etc. at time of myringotomy



Myringotomy

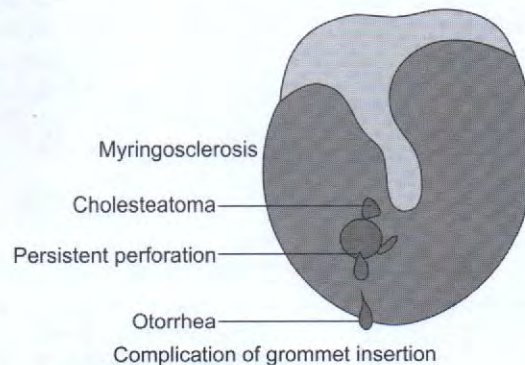


Figure 20: Operative OME

Complications

- Atrophic tympanic membrane and atelctasis of middle ear (due to dissolution of fibrous layer of tympanic membrane)
- Ossicular necrosis (usually long process of incus and sometimes stapes superstructure)
- Tympanosclerosis (fixation of tympanic membrane and ossicle due to deposition of hyalinized collagen)
- Retraction pockets and cholesteatoma (due to invagination of atrophic parts of pars tensa)
- Cholesterol granuloma (due to stasis of secretion in middle ear and mastoid).

6. Glomus tumor.

- Glomus tumor or globus tympanicum is a benign neoplasm of middle ear.

Site of Origin

- From Glomus bodies found on promontory of middle ear along course of tympanic branch of IX nerve.

Pathology

Gross appearance	Microscopic examination
<ul style="list-style-type: none"> • Benign, non-encapsulated but extremely vascular 	<ul style="list-style-type: none"> • Shows masses or sheets of epithelial cells with large nuclei and granular cytoplasm along with abundance of thin walled blood sinusoids with no contractile muscle coat

Spread

Local spread	Metastatic spread
<ul style="list-style-type: none"> • May initially fill middle ear and later perforate through tympanic membrane to present as vascular polyp in external auditory canal • May erode jugular foramen and base of skull • May invade labyrinth, petrous pyramide and mastoid • May invade Eustachian tube to present in nasopharynx • May spread intracranially to middle and posterior cranial fossa 	<ul style="list-style-type: none"> • To lungs and bone (rare) or lymph nodes

Clinical Features

- Seen in age group of 40–50 years
- Females affected five times more than males.

Symptoms	Signs
<ul style="list-style-type: none"> • Slowly progressive hearing loss • Tinnitus (Swishing type) • Profuse bleeding from ear spontaneously or on attempted cleaning (when tumor presents as polyp) • Dizziness or vertigo 	<ul style="list-style-type: none"> • Rising sun appearance of tympanic membrane (red reflex) • Sometimes bluish and bulging tympanic membrane • Positive pulsation sign when ear canal pressure is raised causing tumor to pulsate vigorously and then blanch (Brown's sign) • Conductive deafness • Pulsatile tinnitus, temporarily stopped by carotid pressure • Red, vascular polyp filling meatus which bleeds readily • Facial paralysis • Audible systolic bruit over mastoid

Investigations

Audiological tests	Radiological	Angiography
<ul style="list-style-type: none"> • Reveals conductive deafness 	<ul style="list-style-type: none"> • CT scan shows intact caroticojugular spine (helps to differentiate from glomus jugulare tumor) • MRI gives soft extend to tumor 	<ul style="list-style-type: none"> • Carotid angiography to assess extent of tumor

Treatment

Conservative	Operative
a. Radiotherapy <i>Indications</i> <ul style="list-style-type: none"> – Inoperable tumors – Residual tumors – Recurrences after surgery – Older individuals with surgery contraindicated b. Embolization <i>Indications</i> <ul style="list-style-type: none"> – To reduce tumor vascularity before surgery – Inoperable patients 	a. Excision (treatment of choice) <ul style="list-style-type: none"> – Depending of extent of tumor through <ul style="list-style-type: none"> - Transmeatal approach - Transmastoid approach - Skull-base approach

7. Choanal atresia.

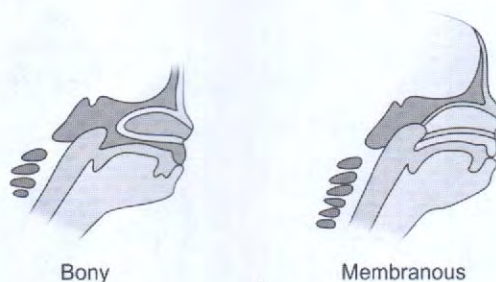
- Congenital anomaly resulting in obstruction of posterior nares.

Etiopathogenesis

Etiology	Pathology
♦ Persistence of bucconasal membrane (usually ruptures at 7th-8th week of gestation)	♦ Bony or membranous partition occluding choana attached to basisphenoid, median pterygoid plate, vomer and hard palate

Types

- Bony (90%)
- Membranous (10%).

**Figure 21:** Choanal atresia—types**Clinical Features**

- Mostly unilateral but may be bilateral
- May be complete or partial.

Symptoms		Signs
Unilateral	Bilateral	
♦ Asymptomatic ♦ Persistent nasal obstruction	♦ Difficulty in breathing in newborn (cyclic asphyxia) ♦ Difficulty in swallowing feeds (suckling asphyxia) ♦ Failure to thrive	♦ Mucoïd discharge with absence of air bubbles ♦ Inability to blow nose on affected side ♦ Complete occlusion of choana with a thin membrane or plate of bone with a dimple in its center ♦ Failure to pass catheter from nose to pharynx

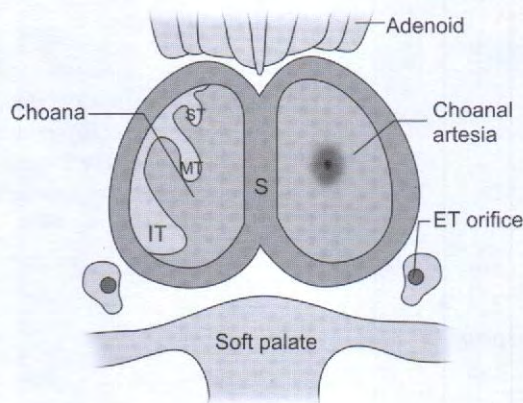


Figure 22: Choanal atresia—signs

Investigations

Dye test	Contrast nasogram	CT scan	Diagnostic nasal endoscopy
Instillation of methylene blue dye in nose and observing its passage into pharynx	Lateral view of neck after instillation of radiopaque dye into nose	Diagnostic	Most useful

Treatment

Conservative	Operative
a. Provision of good oral airway – Using feeding nipple with large hole (McGovern’s technique)	a. Tracheostomy or endotracheal intubation – To establish airway b. Recanalization by – Transnasal route – Simple procedure – Transpalatal route (after 1½ years) – Better exposure

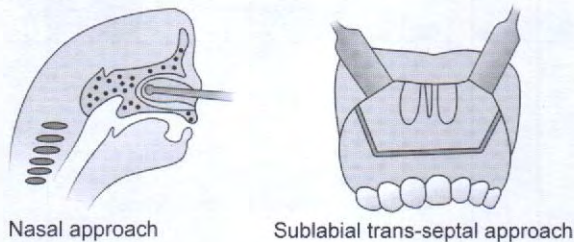


Figure 23: Choanal atesia—recanalization

8. Indications and complications of tonsillectomy.

Refer Question No. 8 December 2010 (RS2).

9. Clinical features and management of acute retropharyngeal abscess.

Refer Question No. 2 December 2009 (RS2).

10. Esophagoscopy.

Refer Question No. 4 June 2012 (RS2).

11. Differential diagnosis of midline neck swellings.

Refer Question No. 8 December 2012 (RS2).

12. Management of atrophic rhinitis.

Refer Question No. 1 December 2014 (RS2).

■ SHORT ANSWERS**13. Stridor.**

Refer Question No. 1 June 2016 (RS2).

14. Waldeyer's ring.

Refer Question No. 3 June 2015 (RS2).

15. Nasal vestibulitis.

Refer Question No. 15 December 2012 (RS2).

16. Fistula test.

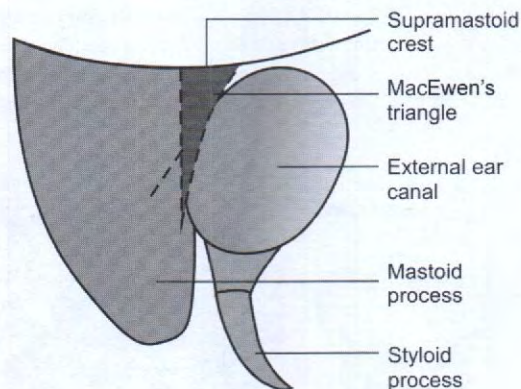
Refer Question No. 3 June 2009 (RS2).

17. MacEwens triangle.

- MacEwen's triangle is triangular area overlying mastoid antrum
- Also called suprameatal triangle.

Boundaries

<i>Anteroinferiorly</i>	<i>Superiorly</i>	<i>Posteriorly</i>
<ul style="list-style-type: none"> ♦ Posterosuperior segment of bony external auditory canal ♦ Suprameatal spine of Henle 	<ul style="list-style-type: none"> ♦ Supramastoid crest (Temporal line) 	<ul style="list-style-type: none"> ♦ Vertical line drawn tangential to posterior margin of external auditory canal

**Figure 24:** MacEwen's triangle**Applied Anatomy**

- MacEwen's triangle is an important anatomical landmark to locate mastoid antrum in mastoid surgery.

18. Rhinophyma.

Refer Question No. 13 June 2011 (RS2).

19. Hearing aid.

Refer Question No. 10 December 2011 (RS2).

20. Otitis externa.

Refer Question No. 15 December 2008 (RS2).

21. Little's area.

Refer Question No. 20 June 2011 (RS2).

22. Pleomorphic adenoma.

- Pleomorphic adenoma is most common benign tumor of parotid salivary gland
- Also called mixed tumor.

Etiology

- Age: Usually seen in 3rd or 4th decade of life
- Sex: More likely in females.

Pathology

<i>Gross appearance</i>	<i>Microscopic appearance</i>
<ul style="list-style-type: none"> ♦ Circumscribed, pseudoencapsulated, rounded, at times multilobulated, firm mass, 2–5 cm in diameter with bosselated surface ♦ Cut surface is gray white and bluish, variegated, semitranslucent, usually solid but occasionally may show small cystic space ♦ Consistency is soft and mucoid 	<ul style="list-style-type: none"> ♦ Characterized by pleomorphic or mixed appearance, i.e. epithelial elements present in matrix of mucoid, myxoid and chondroid tissue with predominance of either <ol style="list-style-type: none"> Epithelial component <ul style="list-style-type: none"> – Consists of cells of ductal or myoepithelial origin arranged in various patterns like ducts, acini, tubules, sheets and strands – Ductal cells are cuboidal or columnar and underlying myoepithelial cells may be polygonal or spindle-shaped resembling smooth muscle cells – Lumen of duct—like structures contain PAS positive epithelial mucin – Focal areas of squamous metaplasia and keratinisation Mesenchymal elements <ul style="list-style-type: none"> – Consists of loose connective tissue and myxoid, mucoid and chondroid matrix simulating cartilage (pseudocartilage) – Matrix of tumor is a product of myoepithelial cells – Small proportion of cases shows true cartilage and even bone

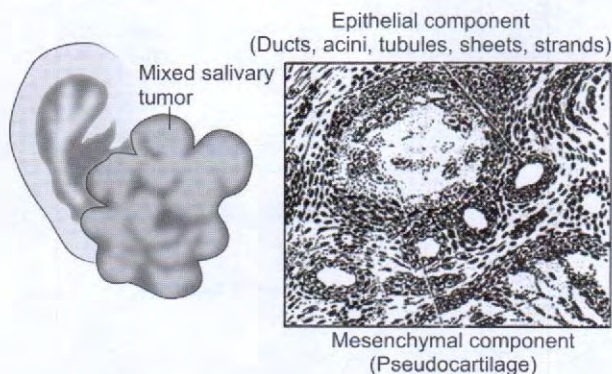


Figure 25: Mixed parotid tumor—pathology

Clinical Features

<i>Symptoms</i>	<i>Signs</i>
<ul style="list-style-type: none"> ♦ Painless swelling in parotid region ♦ Foreign body feeling in throat, dysphagia (if arises from deep lobe) 	<ul style="list-style-type: none"> ♦ Firm, smooth, non-tender, lobulated, mobile slow growing mass in lower part of parotid region ♦ Skin not adhered to underlying mass ♦ Swelling in lateral pharyngeal wall (parapharyngeal mass) in oropharynx with tonsil being pushed medially (if arise from deep lobe) ♦ Sudden increase in size, pain, signs of facial paralysis and fixation to neighboring structures (indication of malignant transformation)

Investigations

- FNAC
- Sialogram
- CT/MRI.

Treatment

Operative (treatment of choice)

	<i>Extracapsular excision</i>	<i>Superficial parotidectomy with preservation (surgery of choice)</i>	<i>Total parotidectomy with or without block dissection of neck</i>
Indications	♦ Very small superficial tumors	<ul style="list-style-type: none"> ♦ Tumor has broken confines ♦ Recurrent tumor after local excision 	♦ Malignant transformation
Disadvantages	♦ High recurrence		

MBBS PHASE III EXAMINATION

JUNE 2014

(Revised Scheme 2 & 3)

■ LONG ESSAYS

- 1. Enumerate the causes of epistaxis. Discuss in detail the etiology and management of epistaxis in a 16-year-old boy.**

Refer Question No. 1 June 2009 (RS2).

- 2. Discuss the etiology, clinical features and management of otosclerosis.**

Refer Question No. 1 December 2010 (RS2).

■ SHORT ESSAYS

- 3. What is stridor? What are its causes?**

Refer Question No. 1 June 2016 (RS2).

- 4. Etiology and management of retropharyngeal abscess.**

Refer Question No. 2 December 2009 (RS2).

- 5. Etiology, clinical features and management of oral submucous fibrosis.**

Refer Question No. 11 June 2010 (RS2).

- 6. Etiology, clinical features and management of rhinoscleroma.**

Refer Question No. 4 December 2013 (RS2).

- 7. Causes and management of atrophic rhinitis.**

Refer Question No. 1 December 2014 (RS2).

- 8. Management of presbycusis.**

- Presbycusis is sensorineural hearing loss associated with physiological aging in ear
- Also called senile deafness.

Predisposing factors

- Hereditary predisposition
- Chronic noise exposure
- Ototoxicity
- Diabetes
- Generalized vascular disease like atherosclerosis.

Types (Pathology)

<i>Sensory type</i>	<i>Neural type</i>	<i>Strial (metabolic) type</i>	<i>Cochlear conductive (mechanical) type</i>
<ul style="list-style-type: none"> ◆ Degeneration of organ of Corti, starting at basal coil and progressing gradually to apex ◆ Affects higher frequencies with good speech discrimination 	<ul style="list-style-type: none"> ◆ Degeneration of cells of spiral ganglion, starting at basal coil and progressing to apex ◆ Also affects neurons of higher auditory pathways ◆ High frequency losses and poor speech discrimination which is out of proportion to pure-tone loss 	<ul style="list-style-type: none"> ◆ Degenerative changes or atrophy of all three layers of stria vascularis in all turns of cochlea ◆ Affects physical and chemical processes of energy production ◆ Runs in families ◆ Flat audiogram with good speech discrimination 	<ul style="list-style-type: none"> ◆ Degeneration of spiral ligament rupture and stiffening of basilar membrane, thus affecting its movements ◆ Descending (sloping) type audiogram

Clinical Features

- Usually manifests at 65 years
- Gradual in onset and progressive in nature
- Bilaterally symmetrical.

Symptoms

- Difficulty in hearing in noisy environment
- Intolerance to loud sounds (positive recruitment phenomenon)
- Tinnitus
- Vertigo
- Distortion of speech.

Investigations

<i>Tuning fork test</i>			<i>Audiogram</i>
Rinne test	Weber test	Absolute bone conduction	Sensorineural hearing loss to high frequency initially and later low frequency also
Positive but short	Lateralized to better ear	Reduced	

Differential Diagnosis

<i>Cochlear otosclerosis</i>	<i>Meniere's disease</i>	<i>Acoustic trauma</i>
<ul style="list-style-type: none"> ◆ Normal bone conduction in lower tone in contrast to marked air conduction loss 	<ul style="list-style-type: none"> ◆ Fluctuating hearing loss ◆ Positive glycerol test 	<ul style="list-style-type: none"> ◆ History of trauma ◆ Sudden dip in audiogram at 4 kHz

Treatment**Conservative**

<i>Supportive</i>	<i>Specific</i>
<ul style="list-style-type: none"> ◆ Avoidance of noise ◆ Curtailment of smoking and stimulant beverages ◆ Speech reading methods ◆ Psychological support 	<ul style="list-style-type: none"> ◆ Vitamin B₁, B₆ and B₁₂ along with iron (to prevent deterioration) ◆ Hearing aids or cochlear implants

9. Otoacoustic emissions.

- Otoacoustic emissions are low intensity sounds produced by movements of outer hair cells of cochlea either spontaneously or in response to an acoustic stimulus
- Also called cochlear echoes or Kemp echoes
- First described by David Kemp in 1978.

Mechanism

- In response to sound, outer hair cells move, generating a mechanical energy inside cochlea that is propagated out (in reverse) via basilar membrane, perilymph, oval window, ossicles, tympanic membrane to external auditory canal, generating a signal
- These vibrations of outer hair cells can be detected in external auditory meatus by using a microphone and computerized techniques.

Types

- Spontaneous otoacoustic emissions
 - Arise spontaneously from outer hair cells
 - Present in normal hearing ear or when hearing loss does not exceed 30 dB
 - May be absent in about 50% of the normal people
 - Inhibited by ototoxic drugs.
- Evoked otoacoustic emission
 - Generated by outer hair cells in response to auditory stimulation
 - Measures only cochlear status and is independent of neural activity and CNS status.

<i>Transient evoked otoacoustic emission</i>	<i>Distortion evoked otoacoustic emission</i>
<ul style="list-style-type: none"> ♦ Provides information over a broad frequency range (500–6000 Hz) that occurs after a brief stimulus ♦ A series of click stimuli are presented at 80–85 dB sound pressure level ♦ Observed in neonatal ears in absence of external and middle ear disorders 	<ul style="list-style-type: none"> ♦ Provides frequency specific information, occurs in response to simultaneous presentation of two pure tones ♦ Two tones are simultaneously presented to the cochlea to produce distortion products ♦ They have been used to test hearing in the range of 1000–8000 Hz ♦ Screening algorithms are robust in neonates and infants and use "DP grams"

Application

- Very useful in "screening of neonates and high-risk infants" for hearing loss
- As hearing test in uncooperative or mentally challenged patients
- Distinguish between cochlear (acoustic trauma and ototoxic drugs) and retrocochlear hearing losses (auditory neuropathy)
- Diagnosing central processing auditory disorders, particularly auditory neuropathy
- Diagnose damage to outer hair cells due to acoustic trauma and ototoxic drugs (detects ototoxic effects earlier than pure-tone audiometry)
- To monitor Ménière's disease
- To detect malingerers
- Provides objective confirmation of cochlear dysfunction in tinnitus.

<i>Advantages</i>	<i>Disadvantages</i>
<ul style="list-style-type: none"> ♦ Noninvasive ♦ Objective (more accurate) ♦ Time saving (takes <3 min) ♦ Needs no preparation ♦ Not interfered by sedation 	<ul style="list-style-type: none"> ♦ Not very useful in mild hearing loss and central auditory pathology ♦ Cannot quantify hearing loss ♦ Not useful in serous otitis media

Significance

- Otoacoustic emissions are a reliable indicator of the integrity of peripheral auditory system
- Measurement of otoacoustic emissions indicates objective measurement of cochlear function
- They are absent in patients suffering from profound sensorineural hearing loss and hearing loss of >40–55 dB
- They do not disappear in VIIIth nerve pathology as cochlear hair cells are normal.

10. What is Killian's dehiscence? Discuss management of pharyngeal pouch.

Killian's Dehiscence

- Killian's dehiscence is a potential space in posterior pharyngeal wall
- Also called gateway of tears (because perforation may occur during esophagoscopy).

Boundaries

Superior	Inferior
♦ Thyropharyngeus (oblique fibers of inferior constrictor)	♦ Cricopharyngeus (transverse fibers of inferior constrictor)

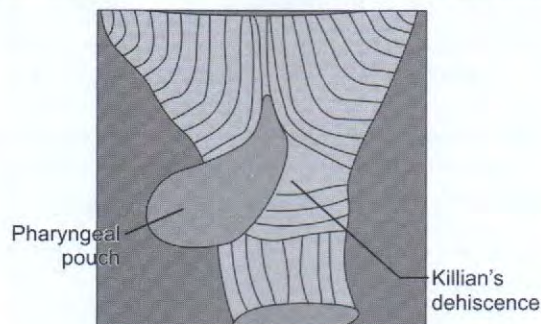


Figure 1: Killian's dehiscence

Applied Anatomy

- Embryologically weak site resulting in herniation of pharyngeal mucosa through it in cases of pharyngeal pouch
- Pharyngeal pouch is more likely to occur when food exerts pressure over this area due to neuromuscular incoordination between propulsive thyropharyngeus (supplied by pharyngeal plexus) and sphincteric cricopharyngeus (supplied by recurrent laryngeal nerve)
- Pharyngeal pouch, thus, formed cannot expand posteriorly due to vertebral column, therefore grows downwards pressing over esophagus resulting in dysphagia.

Pharyngeal Pouch

- Pharyngeal pouch is a pulsion hypopharyngeal diverticulum where hypopharyngeal mucosa herniates through Killian's dehiscence, a weak area between two parts (thyropharyngeal and cricopharyngeal) of inferior constrictor muscle
- Also called as hypopharyngeal diverticulum, Zenker's diverticulum or upper esophageal diverticulæ.

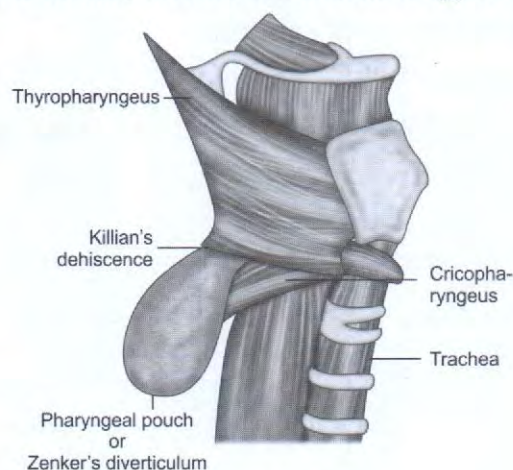


Figure 2: Pharyngeal pouch

Etiopathogenesis

<i>Etiology</i>	<i>Pathology</i>
<ul style="list-style-type: none"> Unknown; may be due to spasm of cricopharyngeal sphincter or its incoordinated contractions during deglutition 	<ul style="list-style-type: none"> Herniation of pouch starts in midline, at first behind esophagus and, then, comes to lie on its left Mouth of sac is wider than pening of esophagus and, thus, food preferentially enters the sac

Clinical Features

- Usually seen after 60 years of age.

<i>Symptoms</i>	<i>Signs</i>
<ul style="list-style-type: none"> Dysphagia (prominent feature), appears after a few swallows when pouch gets filled with food and presses on esophagus Gurgling sound on swallowing Halitosis, transfer dysphagia (difficulty initiating swallowing) Voice changes due to overflow of sac contents into larynx causing chemical irritation and laryngitis or due to pressure of sac on recurrent laryngeal nerve Regurgitation of undigested food at night (when patient is recumbent) causing cough and choking Aspiration pneumonia 	<ul style="list-style-type: none"> Weight loss and malnourishment due to dysphagia Swelling on left side of neck in anterior triangle, which is soft and gurgles on palpation (Boyce sign) Associated with hiatus hernia Rarely malignant transformation (in long-standing cases)

Investigations

- Barium swallow to demonstrate the sac and its size.

Treatment*Operative*

- Diverticulectomy (excision of pouch) or cricopharyngeal myotomy or both—through cervical approach
- Endoscopic division of partition wall between esophagus and pouch (dilatation of cricopharyngeal sphincter)
 - Dohlman's procedure—using diathermy (indicated in high risk or debilitated patients)
 - Laser treatment—using CO₂ laser under operating microscope.

11. Clinical features and management of quinsy.

Refer Question No. 7 June 2011 (RS2).

12. Causes and treatment of deviated nasal septum.

- Deviated nasal septum (DNS) is deviation of cartilage and/or bony framework of nasal septum from midline.

<i>Etiology</i>	<i>Predisposing factors</i>
a. Trauma (especially at birth or during childhood) <ul style="list-style-type: none"> Lateral blow displaces septal cartilage Crushing blow cause buckling, twisting, fracture and duplication with telescoping Compressive birth trauma due to molding, especially during face presentation b. Error in development (commonest cause) <ul style="list-style-type: none"> Unequal growth between palate and base of skull causes buckling of nasal septum Cleft lip, cleft palate and dental anomalies also present with DNS c. Congenital <ul style="list-style-type: none"> Due to abnormal intrauterine posture resulting in compression forces acting on nose and upper jaw d. Secondary <ul style="list-style-type: none"> Due to tumors, mass or polyp in nose 	a. Age <ul style="list-style-type: none"> Usually starts around puberty and reaches full development by 20 years b. Race <ul style="list-style-type: none"> Caucasoid affected more than Negroes c. Heredity <ul style="list-style-type: none"> Affection of several members of a family

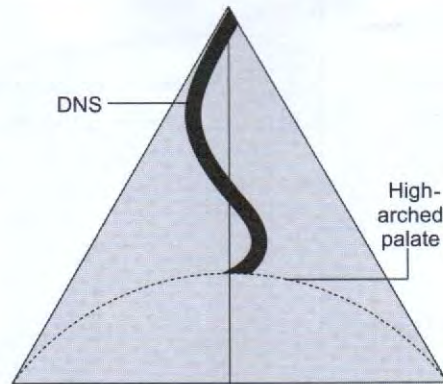


Figure 3: DNS associated with high-arched palate

Pathology (Types)

Deviations	Dislocation	Spurs	Thickening
<ul style="list-style-type: none"> Smooth deflections either upper or lower, anterior or posterior Simply curve to one side (C-shaped deformity) S shaped curving in either vertical or anteroposterior plane (S-shaped deformity) 	<ul style="list-style-type: none"> Dislocation of septal cartilage into one of the nasal chambers 	<ul style="list-style-type: none"> Isolated thickening of bony septum producing shelf like projection at the junction of bone and cartilage 	<ul style="list-style-type: none"> Organized hematoma or overriding of dislocated septal fragments

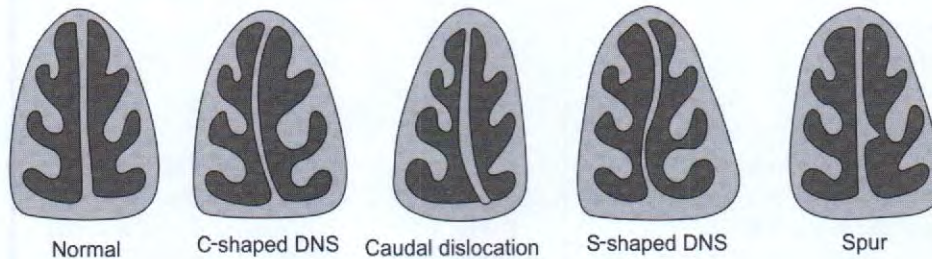


Figure 4: Types of DNS

Clinical Features

- Affects mostly males.

Symptoms	Signs
<p>a. Nasal obstruction (esp. in cases of high septal deviation)</p> <ul style="list-style-type: none"> Either unilateral or bilateral depending upon the type of septal deformity <p>b. Headache</p> <ul style="list-style-type: none"> Pressure headache, especially due to spur pressing on lateral wall Sinusitis headache due to obstruction to drainage of sinuses Vacuum headache due to vacuum created in paranasal sinuses Anterior ethmoidal nerve syndrome <p>c. Bleeding from nose (epistaxis)</p> <ul style="list-style-type: none"> Bleeding from vessels over spur or on removal of crusts (formed due to drying of exposed mucosa to air currents) <p>d. Recurrent attacks of cold</p> <ul style="list-style-type: none"> Due to infection of stagnant nasal secretions <p>e. Decreased smell sensation (hyposmia or anosmia)</p> <ul style="list-style-type: none"> Due to failure of inspired air to reach olfactory region <p>f. External deformity</p> <ul style="list-style-type: none"> Deviation of cartilaginous or bony or both parts of nose 	<p>a. External nose deformity</p> <p>b. Anterior rhinoscopy</p> <ul style="list-style-type: none"> Deviated or deformed nasal septum or spurs Secondary hypertrophy of turbinates <p>c. Cottle test</p> <ul style="list-style-type: none"> Pulling of cheek outwards at nasofacial crease improves nasal patency at valve area

Investigations

<i>Radiological</i>	<i>Diagnostic nasal endoscopy</i>
<ul style="list-style-type: none"> ♦ Water's view or Caldwell view to look for haziness of sinuses and alignment of septum and size of turbinates ♦ CT scan to assess pathology, contact areas, status of osteomeatal complex and presence of concha bullosa 	<ul style="list-style-type: none"> ♦ For precise identification of pathological site, anatomical variation and abnormalities of septum and lateral wall

Differential Diagnosis

<i>Hypertrophic turbinates</i>	<i>Polyps</i>	<i>Septal haematoma</i>
<ul style="list-style-type: none"> ♦ Soft mucosa on turbinates ♦ Sensation of touching bone on deep palpation by a probe ♦ Shrinking of hypertrophic turbinates by decongestant drops 	<ul style="list-style-type: none"> ♦ Soft, pearly white ♦ Insensitive to touch 	<ul style="list-style-type: none"> ♦ Soft watch-glass like swelling on both sides

Treatment

	<i>Conservative</i>	<i>Operative</i>
Indications	<ul style="list-style-type: none"> ♦ Asymptomatic cases 	<ul style="list-style-type: none"> ♦ Mechanical nasal obstruction ♦ Persistent and recurrent symptoms
Techniques	<ul style="list-style-type: none"> ♦ Symptomatic for epistaxis, rhinitis, etc. 	<ul style="list-style-type: none"> ♦ Submucous resection ♦ Septoplasty

a. Submucous resection

- Treatment of choice.

<i>Indications</i>	<i>Contraindications</i>
<ul style="list-style-type: none"> ♦ Symptomatic DNS with recurrent headache ♦ DNS with recurrent sinusitis or recurrent otitis media ♦ Recurrent epistaxis due to septal spur ♦ Cosmetic correction of external nasal deformity (as component of septorhinoplasty) ♦ Approach to cranial fossa for hypophysectomy (transseptal transsphenoidal approach) or for vidian neurectomy (transseptal approach) ♦ Procuring septal cartilage for grafting in tympanoplasty or rhinoplasty 	<ul style="list-style-type: none"> ♦ Age below 17 years (interferes with development of facial bones) ♦ Acute respiratory infection ♦ Bleeding diathesis ♦ Untreated diabetes or hypertension

Anesthesia

- Local infiltrative anesthesia in subchondrial plane with 2% xylocaine and 1:1,00,000 adrenaline
- Local topical anesthesia with 4% xylocaine and 1:1,00,000 adrenaline.

Position

- Reclining position with raised head end of table.

Incision

- Curvilinear incision with forward convexity at mucocutaneous junction on left side of septum.

Procedure

- After incision, elevate mucoperichondrial and periosteal flap using an elevator
- Incise cartilage just posterior to incision with precaution to avoid cutting opposite mucoperichondrium (thus, avoids incision on mucoperichondrium on both sides at identical spots to prevent septal perforation)
- Pass an elevator through cartilage incision to elevate mucoperichondrial and periosteal flap
- Remove cartilage using Ballenger swivel knife but preserving 1cm wide strip of cartilage along dorsal and caudal border of septum (to prevent collapse of bridge of nose or retraction of columella)

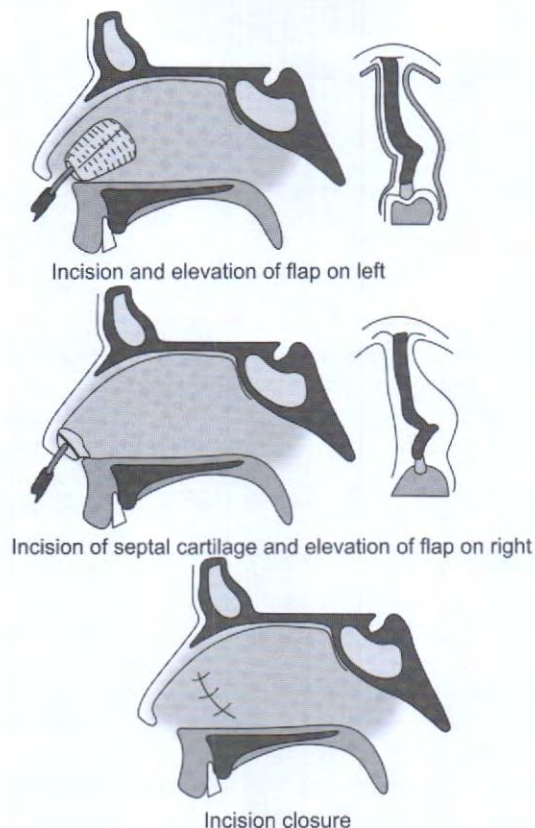


Figure 5: SMR—procedure

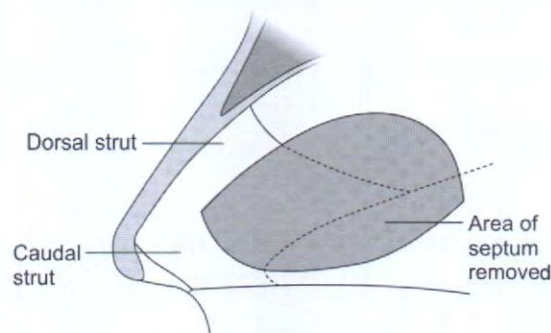


Figure 6: SMR—area of septum removed

- Remove bone using Luc's forceps or bony spurs or ridges using gouge and hammer
- Apply catgut or silk sutures on first mucoperichondrial incision
- Pack nose with antibiotic or liquid paraffin soaked ribbon gauze.

Postoperative care

- Place patient in semi-sitting position (to prevent oozing of blood)
- Soft diet for 2 days (to prevent bleeding due to mastication)
- Nasal pack removed after 24 hours
- Stitches removed after 5–6 days
- Antibiotics for 5–6 days
- Analgesics (for pain, if any)
- Decongestant nasal drops and steam inhalation for 5–6 days (after removal of nasal packs).

Complications**Immediate**

- i. Primary hemorrhage
 - Controlled by adrenaline pack or bony hemostat, packing, etc.
- ii. Damage to surrounding structures
- iii. Cerebrospinal fluid rhinorrhea (if cribriform plate damaged)
- iv. Anesthetic complications

Delayed

	<i>Etiology</i>	<i>Treatment</i>
i. Hemorrhage		
Reactionary hemorrhage	♦ Within 48 hours due to waning of adrenaline effect or postoperative rise in blood pressure	♦ Firm repacking of nose
Secondary hemorrhage	♦ Infection	♦ Changing antibiotic ♦ Repacking of nose and coagulants ♦ Blood transfusion, if necessary
ii. Septal hematoma	♦ Iatrogenic trauma	♦ Wide bore needle aspiration ♦ Incision and drainage ♦ Antibiotics and analgesics
iii. Septal abscess	♦ Infection of septal hematoma	♦ Antibiotics and analgesics ♦ Wide bore needle aspiration ♦ Incision and drainage
iv. Septal perforation	♦ Iatrogenic ♦ Septal hematoma ♦ Septal abscess	♦ Septal buttons ♦ Surgical closure
v. Synechiae	♦ Adhesions between injured septal mucosal flap and turbinate at identical spots	♦ No treatment if asymptomatic ♦ Severance of adhesions and inserting polyethylene sticks for 2 weeks to avoid contact of raw edges
vi. Depression of bridge	♦ Excessive resection of septum at dorsal end	♦ Rhinoplasty
vii. Retraction of columella	♦ Excessive resection of septum at caudal end	♦ Rhinoplasty
viii. Persistence of deviation		
ix. Flapping nasal septum	♦ Excessive removal of septal structure, resulting in weak septum yielding to inspiratory negative pressure in nose	♦ Self correcting due to fibrosis
x. Toxic shock syndrome		

b. Septoplasty

- Conservative approach preserving as much as possible of septal framework.

<i>Indications</i>	<i>Contraindications</i>
<ul style="list-style-type: none"> ♦ Symptomatic DNS in children ♦ Recurrent epistaxis due to septal spur ♦ Cosmetic correction of external nasal deformity (as component of septorhinoplasty) ♦ Approach to cranial fossa for hypophysectomy (transseptal transsphenoidal approach) 	<ul style="list-style-type: none"> ♦ Acute nasal or sinus infection ♦ Bleeding diathesis ♦ Untreated diabetes or hypertension

Anesthesia

- Local infiltrative anesthesia in subchondrial plane with 1% xylocaine and 1:1,00,000 adrenaline
- Local topical anesthesia with 4% xylocaine
- General anesthesia for uncooperative patients.

Position

- Reclining position with raised head end of table.

Incision

<i>Killian's incision</i>	<i>Freer's incision</i>
♦ Slightly curvilinear incision at 2–3 mm above caudal end of septal cartilage on concave side	♦ Transfixation or hemifixation incision for caudal dislocation

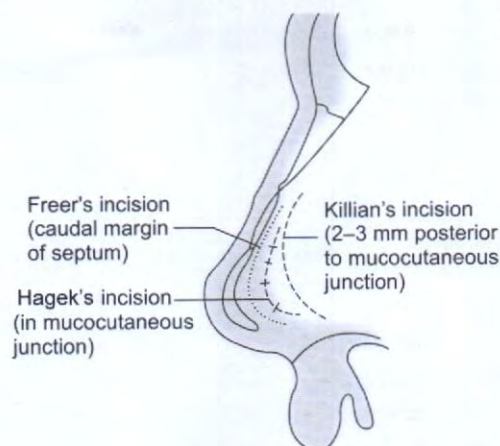


Figure 7: Incision for septoplasty

Procedure

- After incision, elevate mucoperichondrial flap using an elevator creating anterior tunnel
- Elevate mucoperiosteum over maxillary crest creating inferior tunnel
- Join both tunnels by raw dissection
- Separate septal cartilage from vomer and ethmoidal plate
- Raise mucoperiosteal flap on opposite side of septum
- Remove maxillary crest to realign septal cartilage
- Bony septum corrected by removing deformed bony parts
- Cartilage septum corrected by scoring on concave side, cross hatching or morcelizing, shaving or wedge incision
- Apply catgut or silk sutures (transseptal)
- Pack nose with antibiotic or liquid paraffin soaked ribbon gauze.

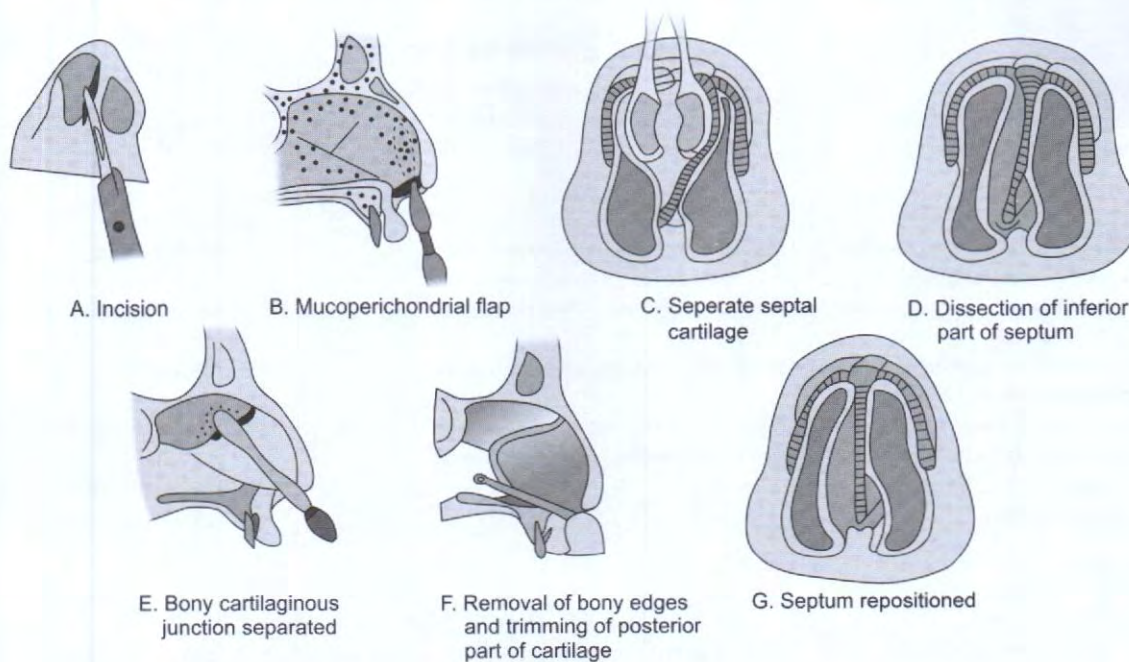


Figure 8: Septoplasty—procedure

Postoperative care

- ◆ Place patient in semi-sitting position (to prevent oozing of blood)
- ◆ Soft diet for 2 days (to prevent bleeding due to mastication)
- ◆ Nasal pack removed after 24 hours
- ◆ Stitches removed after 5–6 days
- ◆ Antibiotics for 5–6 days
- ◆ Analgesics (for pain, if any)
- ◆ Decongestant nasal drops and steam inhalation for 5–6 days (after removal of nasal packs)

Complications

- ◆ Bleeding
- ◆ Septal hematoma
- ◆ Septal abscess
- ◆ Septal perforation
- ◆ Persistence of deviation
- ◆ External nasal deformity

Complications (of DNS)

a. Recurrent sinusitis	Due to obstruction to drainage of sinuses
b. Middle ear infections	Due to recurrent upper respiratory tract infections or forcible blowing of nose
c. Mouth breathing	Leading to recurrent upper respiratory infections
d. Asthma	Due to triggering of bronchospasm
e. Atrophic rhinitis	On roomy side of nose

SHORT ANSWERS**13. Adenoid facies.**

Refer Question No. 7 June 2016 (RS2).

14. Management of vocal nodule.

Refer Question No. 9 June 2016 (RS2).

15. Premalignant lesions of larynx.

- Precancerous lesions are those conditions which are not histologically malignant, but for which there is satisfactory proof that cancer occurs in them with higher incidence than corresponding normal tissues.

Examples

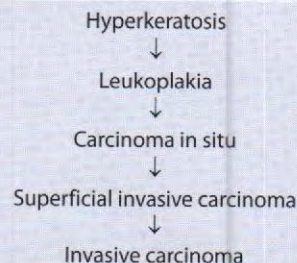
- ◆ Hyperkeratosis
- ◆ Leukoplakia
- ◆ Papilloma of larynx

Predisposing factors

- ◆ Chronic laryngeal irritation
- ◆ Chronic laryngitis
- ◆ Radiation burns

Pathology

- ◆ Hyperkeratosis is a thickening of superficial layers of the epithelium. The area may appear as having warty or grayish papillomatous appearance. Histologically, there is thickening of the stratum corneum with little or no inflammatory reaction in the underlying tissues
- ◆ Leukoplakia appears as a white patch usually on the cords and may look like a small prominent patch or a nodular plaque
- ◆ Solitary papilloma appears as pink warty growth of varying size in the anterior commissure arising from edge of the vocal cord in the anterior part and may be pedunculated
- ◆ Cellular atypia is evident

Pathogenesis**Clinical Features**

- Hoarseness of voice
- Involves upper surface of either one or both vocal cords but does not affect mobility of cord
- Lesions may be single or multiple.

<i>Treatment</i>	<i>Prevention</i>
<ul style="list-style-type: none"> ♦ Microlaryngoscopy stripping of vocal cords 	<ul style="list-style-type: none"> ♦ Smoking cessation ♦ Reduced alcohol drinking ♦ Toxin free work environment ♦ Healthy diet

16. Rhinitis Medicamentosa.

Refer Question No. 19 June 2009 (RS2).

17. Theories of hearing.

Refer Question No. 1 December 2007 (RS2).

18. Management of Bell's palsy.

Refer Question No. 4 December 2012 (RS2).

19. Causes of oroantral fistula.

- Oroantral fistula (orodental fistula) is a communicating track between maxillary antrum and oral cavity.

Location

- Upper alveolus or gingivolabial sulcus.

Etiology

- Dental extraction of upper 2nd premolar and molars (1st and sometimes 2nd and 3rd)—roots of these teeth closely related to cavity of maxillary antrum (most common cause)
- Postoperative complication of Caldwell-Luc operation—sublabial incision may fail to heal
- Carcinoma of maxilla—erosion of floor of maxillary antrum
- Penetrating injuries and fractures of maxilla
- Infection of maxilla—osteitis and syphilis.

Causes of oroantral fistula: DEC Chill Is irritating

Dental Extraction
Caldwell Luc - complication
Carcinoma of maxilla
Injuries of maxilla
Infections of maxilla

Predisposing Factors

- Apical teeth abscess (important predisposing factor).

Clinical Features

<i>Symptoms</i>	<i>Signs</i>
<ul style="list-style-type: none"> ♦ Nasal regurgitation—semisolid food and liquid pass through fistula and come into antrum and then to nose ♦ Foul smelling discharge from fistula and nose—due to infection of maxillary antrum ♦ Inability to create positive and negative pressure in oral cavity—difficulty in blowing musical instruments or suck through a straw 	<ul style="list-style-type: none"> ♦ Probing through fistulous tract from oral cavity into antrum

Treatment

<i>Recent fistula</i>	<i>Chronic/large fistula</i>	<i>Caldwell-Luc operation</i>	<i>Carcinoma</i>
<ul style="list-style-type: none"> ♦ Suturing of gum margins and a course of antibiotic after dental extraction 	<ul style="list-style-type: none"> ♦ Conservative treatment of maxillary sinusitis with antibiotics and repeated antral irrigation ♦ Surgical repair by excision of fistulous track, smoothening of edges of fistula and closure with palatal or buccal flap 	<ul style="list-style-type: none"> ♦ Done to remove retained tooth root, foreign body and diseased mucosa ♦ Provides for nasoantral window for free drainage 	<ul style="list-style-type: none"> ♦ Closure by a dental obturator

20. Management of nasal myiasis.

Refer Question No. 17 December 2007 (RS2).

21. Uses of Lasers in ENT.

Refer Question No. 20 December 2010 (RS2).

22. Management of reactionary hemorrhage following tonsillectomy.

Refer Question No. 8 December 2010 (RS2).

MBBS PHASE III EXAMINATION

DECEMBER 2014

(Revised Scheme 2 & 3)

LONG ESSAYS

1. Describe the etiology, clinical features and management of atrophic rhinitis.

- Atrophic rhinitis is chronic inflammation of nose characterized by progressive atrophy of nasal mucosa and turbinate bones
- Also called ozaena (due to associated foul smell from nose).

Etiology

Primary atrophic rhinitis (HERNIA)	Secondary atrophic rhinitis
a. Hereditary factors <ul style="list-style-type: none">– Involvement of more than one family member	a. Specific infections <ul style="list-style-type: none">– Syphilis, lupus, leprosy and rhinoscleroma
b. Endocrinal disturbances <ul style="list-style-type: none">– Due to fact that disease seen predominantly in females at puberty and tend to cease after menopause	b. Iatrogenic <ul style="list-style-type: none">– Excessive surgical removal of turbinates– Radiotherapy to nose
c. Racial factors <ul style="list-style-type: none">– Affects Caucasoid and Mongoloids more than Negroes	c. Infections of paranasal sinuses <ul style="list-style-type: none">– Chronic sinusitis
d. Nutritional deficiency <ul style="list-style-type: none">– Due to deficiency of vitamin A, D or iron	
e. Infection <ul style="list-style-type: none">– Klebsiella ozaenae, diphtheroids, Proteus vulgaris, E. coli, staphylococci and streptococci isolated from cultures but considered to be secondary invaders	
f. Autoimmune process <ul style="list-style-type: none">– Triggering of antigenicity of nasal mucosa by viral infection or other unspecified agent may result in destructive response	

Pathology

a. Nasal mucosa and submucosa	<ul style="list-style-type: none">♦ Loss and replacement of ciliated columnar epithelium by stratified squamous epithelium (squamous metaplasia)♦ Atrophy of seromucinous glands, venous blood sinusoids♦ Pale mucosa with thick scanty secretions♦ Submucous infiltration by round cells
b. Turbinates	<ul style="list-style-type: none">♦ Atrophied producing wide roomy nasal cavity
c. Blood vessels	<ul style="list-style-type: none">♦ Obliterative endarteritis of arteries in mucosa, periosteum and bone
d. Nerves	<ul style="list-style-type: none">♦ Atrophy of sensory nerve endings as well as olfactory nerves

Clinical Features

- Seen predominately in females
- Starts around puberty
- Usually bilateral but may be unilateral.

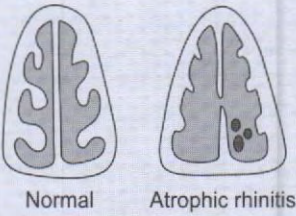
Symptoms	Signs
<p>a. Foul smell from nose (foetor)</p> <ul style="list-style-type: none"> – A social problem keeping friends and relatives away from patient <p>b. Anosmia</p> <ul style="list-style-type: none"> – Due to atrophy of olfactory nerves – Called merciful anosmia because sparing patient of putrid smell of his own nose <p>c. Nasal obstruction</p> <ul style="list-style-type: none"> – In spite of unduly wide nasal chambers – Due to atrophy of sensory nerve endings, patient does not feel airflow in nose or large crusts blocking nose <p>d. Dryness</p> <ul style="list-style-type: none"> – Due to constant air current through roomy wide nasal cavities dry up nasal secretions <p>e. Crusting</p> <ul style="list-style-type: none"> – Nasal cavity filled with large, foul smelling crusts <p>f. Epistaxis</p> <ul style="list-style-type: none"> – On removal of crusts 	<p>a. External nose</p> <ul style="list-style-type: none"> – Saddle deformity of nose (due to atrophy of nasal septum) – Broadened nose with widened nostrils <p>b. Anterior rhinoscopy</p> <ul style="list-style-type: none"> – Pale and atrophic nasal mucosa – Greenish or grayish black dry crusts covering turbinates and septum – Bleeding on attempt to remove them – Atrophy of turbinates reducing them to mere ridges – Easily visible posterior wall of nasopharynx (due to atrophy of turbinates) – Septal perforation and dermatitis of nasal vestibule <p>c. Posterior rhinoscopy</p> <ul style="list-style-type: none"> – Crusting <p>d. Associated features</p> <ul style="list-style-type: none"> – Small and underdeveloped paranasal sinuses with thick walls – Atrophic pharyngitis (dry pharyngeal mucosa, glazed with crusts) or atrophic laryngitis (cough and hoarseness of voice)
	 <p>Normal Atrophic rhinitis</p>

Figure 1: Normal and atrophic rhinitis

Investigations

Radiology (X-ray)	Laboratory investigations	Nasal smear and biopsy
<ul style="list-style-type: none"> ♦ Paranasal sinuses appear opaque with thick walls ♦ May reveal primary or secondary sinusitis 	<ul style="list-style-type: none"> ♦ To rule out underlying etiology (VDRL for syphilis, mountex test for tuberculosis, etc.) 	<ul style="list-style-type: none"> ♦ To confirm diagnosis

Differential Diagnosis

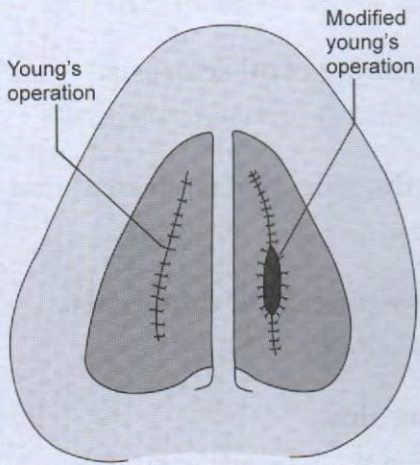
Rhinitis sicca	Rhinoscleroma	Rhinolith or foreign body	Sinusitis
<ul style="list-style-type: none"> ♦ Crusting only in anterior part ♦ No foul smell 	<ul style="list-style-type: none"> ♦ Atrophic changes in early stage but mucosa is pink 	<ul style="list-style-type: none"> ♦ Unilateral and hard to feel 	<ul style="list-style-type: none"> ♦ Foul smell (if of dental origin) ♦ No crusting

Treatment

Conservative	Operative
<p>Principle</p> <ul style="list-style-type: none"> ♦ Complete cure of disease is not yet possible thus treatment aims at maintaining nasal hygiene by removal of crusts and associated putrefying smell and further check crust formation 	<ul style="list-style-type: none"> ♦ Narrow internal dimension of nose or provide rest to nose

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Conservative	Operative
<p>Techniques</p> <p>a. Nasal irrigation and removal of crusts</p> <ul style="list-style-type: none"> Nasal irrigation using warm normal saline or an alkaline solution (1 part sodium bicarbonate to loosen crusts, 1 part sodium baborate acts as antiseptic and 2 part sodium chloride to provide isotonicity; 1 tablespoon in 280 mL of water) Loosen crusts and remove thick tenacious discharge Initially 2–3 times a day but later once in 2–3 days Hard crusts are removed mechanically with forceps or suction after nasal irrigation <p>b. 25% glucose in glycerine</p> <ul style="list-style-type: none"> Paint nose with 25% glucose in glycerine after nasal irrigation Glucose ferments into lactic acid and inhibits growth of proteolytic organisms responsible for foul smell <p>c. Local antibiotics</p> <ul style="list-style-type: none"> Chloramphenicol or streptomycin nasal drops Spraying or painting nose with Kemicetine antiozena solution (chloromycetin, estradiol and vitamin D₂) to eliminate secondary infection <p>d. Estradiol spray</p> <ul style="list-style-type: none"> Increases vascularity of nasal mucosa and helps in regeneration of seromucinous glands <p>e. Placental extract</p> <ul style="list-style-type: none"> Injected submucosally in nose weekly Act as local biogenic stimulant and help in regeneration of epithelium and glandular tissue <p>f. Streptomycin</p> <ul style="list-style-type: none"> 1 g/day for 10 days IM effective against <i>Klebsiella</i> Reduces crusting and odor <p>g. Potassium iodide (orally)</p> <ul style="list-style-type: none"> Promotes and liquefies nasal secretion 	<p>a. Young's operation or modified Young's operation</p> <ul style="list-style-type: none"> Involves closing of both nostrils completely (partially in modified technique) just within nasal vestibule by raising mucosal flaps from inside of nasal vestibules and suturing them by Gadre's modification for 6 months to 1 year  <p>Figure 2: Young's operation</p> <p>Advantages</p> <ul style="list-style-type: none"> Provides prolonged rest to nose thus help reverts nasal mucosa back to normal Prevents drying of nose by air currents thus reduces crusting <p>Disadvantages</p> <ul style="list-style-type: none"> Patient has to breathe through mouth Snoring at night <p>b. Narrowing of nasal cavities</p> <p>Principle</p> <ul style="list-style-type: none"> Wide nasal cavities in atrophic rhinitis predispose to drying of secretions leading to crusting due to air currents thus narrowing of nasal cavities help prevent this <p>Techniques</p> <ul style="list-style-type: none"> Submucosal injection of Teflon paste Insertion of fat, cartilage, bone or Teflon strip under mucoperiosteum of floor and lateral wall of nose and mucoperichondrium of septum Section and medial displacement of lateral wall of nose <p>c. Transfer of Stensen's duct to maxillary sinus</p> <ul style="list-style-type: none"> Helps increasing moisture of nose <p>d. Sympathetic block or cervical sympathectomy</p> <ul style="list-style-type: none"> Improves blood supply to nose
<p>Complications</p> <ul style="list-style-type: none"> Sinusitis Otitis media Atrophic pharyngitis and atrophic laryngitis Psychological complications Maggots 	<p>Prognosis</p> <ul style="list-style-type: none"> Disease persists for years but has tendency to recover spontaneously in after 45 years

2. Describe the etiology, clinical features and management of acute retropharyngeal abscess.

Refer Question No. 2 December 2009 (RS2).

■ SHORT ESSAYS

3. Sleep apnea syndrome.

Refer Question No. 10 June 2010 (RS2).

4. Management of otosclerosis.

Refer Question No. 1 December 2010 (RS2).

5. Etiopathogenesis of cholesteatoma.

Refer Question No. 1 June 2013 (RS2).

6. Cerebrospinal fluid rhinorrhea.

Refer Question No. 6 June 2010 (RS2).

7. Hearing aids.

Refer Question No. 10 December 2011 (RS2).

8. Indications and complications of adenoidectomy.

Refer Question No. 7 June 2016 (RS2).

9. Plummer-Vinson syndrome.

- Syndrome characterized by gradually progressive dysphagia to solids with microcytic hypochromic anemia and chronic pharyngoesophagitis
- Also called Peterson-Brown-Kelly syndrome, sideropenic dysphagia.

Etiopathogenesis

<i>Etiology</i>	<i>Pathology</i>
<ul style="list-style-type: none"> ♦ Iron deficiency anemia <ul style="list-style-type: none"> – May cause it or may result due it ♦ Geography <ul style="list-style-type: none"> – Common in people of Scandinavian origin 	<ul style="list-style-type: none"> ♦ Thinning of mucosa of upper alimentary tract with disappearance of rete pegs ♦ Degeneration of esophageal musculature ♦ Submucosal fibrosis and atrophy of mucosa forming web at pharyngoesophageal junction

Clinical Features

- Predominantly affects females aged 40 years.

<i>Symptoms</i>	<i>Signs</i>
<ul style="list-style-type: none"> ♦ Difficulty in swallowing (dysphagia) for solids initially followed by liquids ♦ Lump in throat ♦ Painful oral ulcers 	<ul style="list-style-type: none"> ♦ Pallor (due to iron deficiency anemia) ♦ Glottitis ♦ Angular stomatitis ♦ Koilnychia (spooning of nails) ♦ Splenomegaly

Investigations

<i>Blood investigations</i>	<i>Gastric analysis</i>	<i>Barium meal or swallow</i>	<i>Esophagoscopy</i>
<ul style="list-style-type: none"> ♦ Reveals iron deficiency anemia ♦ ↓ iron levels ♦ ↑ iron binding capacity 	<ul style="list-style-type: none"> ♦ Reveals achlorhydria 	<ul style="list-style-type: none"> ♦ Shows a web in postcricoid region or narrowing of hypopharynx 	<ul style="list-style-type: none"> ♦ Demonstrates web formation in hypopharynx and detects malignant transformation

Treatment

<i>Conservative</i>	<i>Operative</i>
<ul style="list-style-type: none"> ♦ Correction of anemia with oral/parenteral iron and vitamin B₁₂ and B₆ 	<ul style="list-style-type: none"> ♦ Esophageal bougies to dilate webbed area ♦ Endoscopic dilatation of postcricoid region

Complications

- Postcricoid carcinoma (10%)
- Carcinoma of tongue, buccal mucosa, pharynx, esophagus and stomach.

10. Parotid neoplasms.

- 80% of salivary neoplasms are parotid neoplasms, 80% are benign and 20% are pleomorphic (Parotid—80%).

Classification

<i>Benign</i>		<i>Malignant</i>	
<i>Epithelial</i>	<i>Mesenchymal</i>	<i>Epithelial</i>	<i>Mesenchymal</i>
<ul style="list-style-type: none"> ♦ Pleomorphic adenoma ♦ Warthin's lymphoma ♦ Oxyphil adenoma 	<ul style="list-style-type: none"> ♦ Hemangioma ♦ Lymphangioma ♦ Lipoma ♦ Neurofibroma 	<ul style="list-style-type: none"> ♦ Mucoepidermoid carcinoma ♦ Adenoid cystic carcinoma ♦ Acinic cell carcinoma ♦ Malignant mixed tumor ♦ Squamous cell carcinoma 	<ul style="list-style-type: none"> ♦ Lymphoma ♦ Sarcoma

Etiology

a. Radiation	♦ Exposure to ionizing radiation increases risk of both benign and malignant tumors (especially mucoepidermoid carcinoma)
b. Viral	♦ Epstein-Barr virus is associated with lymphoepithelial carcinoma
c. Smoking	♦ Warthin's tumor is associated with cigarette smoking
d. Occupational factors	♦ Increased risk is seen with exposure to silica dust, nickel alloys and nitrosamines (rubber workers) and use of kerosene as cooking fuel
e. Hormonal factors	♦ High risk in women with a history of early menarche and nulliparity
f. Dietary factors	♦ Two fold risk seen on consumption of vegetables preserved in salt
g. Genetic factors	i. Allelic loss <ul style="list-style-type: none"> – Loss at 12q in pleomorphic adenomas and multiple losses at 9p, 3p and 17p in carcinoma ex-pleomorphic adenoma – Loss of heterozygosity occurs at 8q, 12q and 17p in carcinoma expleomorphic adenoma (17p in high disease stage and increased proliferative rate) ii. Monosomy and polysomy <ul style="list-style-type: none"> – Monosomy of chromosome 17 and polysomy of chromosomes 3 and 17 is higher in adenoid cystic carcinoma iii. Structural rearrangement <ul style="list-style-type: none"> – Cytogenetically, monoclonal and high frequency of tumor-specific chromosome bands abnormalities 3p21, 8q12 and 12q14-15 are seen in pleomorphic adenomas – Ectopic expression of the PLAG1 gene occurs in pleomorphic adenomas with 8q12 aberrations

Features	Treatment
Benign tumors	
a. Pleomorphic adenoma <ul style="list-style-type: none"> – Usually seen in 3rd or 4th decade of life and More likely in females – Painless, firm, smooth, non-tender, lobulated, mobile slow growing mass in lower part of parotid region – Foreign body feeling in throat, dysphagia (if arises from deep lobe) – Skin not adhered to underlying mass – Swelling in lateral pharyngeal wall (parapharyngeal mass) in oropharynx with tonsil being pushed medially (if arise from deep lobe) – Sudden increase in size, pain, signs of facial paralysis and fixation to neighbouring structures (indication of malignant transformation) 	<ul style="list-style-type: none"> ♦ Superficial parotidectomy with preservation (surgery of choice) ♦ Extracapsular excision (very small superficial tumors) ♦ Total parotidectomy with or without block dissection of neck (malignant transformation)
b. Warthin's lymphoma (Adenolymphoma) <ul style="list-style-type: none"> – Commonly seen in 5th to 7th decade of life with 5 times male preponderance, involving tail of parotid unilaterally or bilaterally (10%) – Presents as multiple, rounded encapsulated tumours, at time cystic with mucoid or brownish fluid – Epithelial and lymphoid elements are seen on microscopic examination 	<ul style="list-style-type: none"> ♦ Enucleation through superficial parotidectomy
c. Oncocytoma (Oncocytoma) <ul style="list-style-type: none"> – Rare tumours arising from acidophilic cells called oncocytes, seen mostly in elderly – Involve superficial lobe of parotid and usually do not grow larger than 5cm – Benign oncocytomas are cystic whereas malignant are solid 	<ul style="list-style-type: none"> ♦ Superficial parotidectomy
d. Haemangiomas <ul style="list-style-type: none"> – Most common benign tumours of parotid in children, predominantly affecting females – Most are discovered at birth, growing rapidly in neonatal period to involute spontaneously – Soft and painless swelling which increase in size with crying or straining – Overlying skin shows bluish discoloration 	<ul style="list-style-type: none"> ♦ Surgical excision if they do not regress spontaneously
e. Lymphangioma <ul style="list-style-type: none"> – Less common tumours involving submandibular gland along with parotids – Feel soft and cystic 	<ul style="list-style-type: none"> ♦ Surgical excision if they do not regress spontaneously
Malignant tumors	
a. Mucoepidermoid carcinoma <ul style="list-style-type: none"> – Slow growing tumor that is known to invade facial nerve – Shows areas of mucin-producing cells and squamous cells on microscopy (hence the name) – Higher proportion of epidermoid element, more malignant is tumor – Low grade <ul style="list-style-type: none"> - Good prognosis - More common in children – High grade <ul style="list-style-type: none"> - More aggressive and poor prognosis 	<ul style="list-style-type: none"> ♦ Low grade <ul style="list-style-type: none"> – Treated by superficial or total parotidectomy, preserving facial nerve ♦ High grade <ul style="list-style-type: none"> – Treated by total parotidectomy, sacrificing facial nerve (if invaded)
b. Adenoid cystic carcinoma (Cylindroma) <ul style="list-style-type: none"> – Slow-growing tumor but infiltrates widely into tissue planes, muscles, perineural spaces and lymphatics and thus causes pain and VIIth nerve paralysis – Local recurrences after surgical excision is common – Locally metastasize to lymph nodes and distant to lung, brain and bone 	<ul style="list-style-type: none"> ♦ Radical parotidectomy followed by postoperative radiation (if margins are not free of tumor) ♦ Radical neck dissection if nodal metastases is present
c. Acinic cell carcinoma <ul style="list-style-type: none"> – Low-grade tumor – Presents as a small, firm, movable and encapsulated tumor, sometimes bilateral – Metastases are rare 	<ul style="list-style-type: none"> ♦ Superficial or total parotidectomy

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Features	Treatment
d. Malignant mixed tumor	
<ul style="list-style-type: none"> Appears in two varieties, i.e. (i) develops in pre-existing benign mixed tumour or (ii) develops "de novo" Rapid growth and pain in a benign tumor should always arouse a suspicion of malignant change 	♦ Radical parotidectomy
e. Squamous cell carcinoma	
<ul style="list-style-type: none"> Rapidly growing tumor that infiltrates, causes pain and ulcerates through skin Can metastasize to neck nodes 	♦ Radical parotidectomy followed by postoperative radiation ♦ Radical neck dissection if nodal metastases is present

Staging (American Joint Committee on Cancer (AJCC) Cancer Staging and UICC TNM classification of malignant tumors of major salivary glands)

Tumor		Node involvement		Metastases	
Tx	Primary tumor cannot be assessed	Nx	Regional lymph nodes cannot be assessed	Mx	Distant metastasis cannot be assessed
T0	No evidence of primary tumor	N0	No regional lymph node metastasis	M0	No distant metastasis
T1	Tumor ≤ 2 cm size	N1	Single ipsilateral lymph node ≤ 3 cm	M1	Distant metastasis
T2	Tumor 2–4 cm	N2a	Single ipsilateral lymph node 3–6 cm		
T3	Tumor > 4 cm or extraparenchymal extension	N2b	Multiple ipsilateral lymph nodes < 6 cm		
T4a	Tumor invades skin, mandible, ear canal, or facial nerve	N2c	Bilateral or contralateral lymph nodes < 6 cm		
T4b	Tumor invades skull base, pterygoid plates, or encases carotid artery	N3	Metastasis in lymph node > 6 cm		

Grading

Stage I	T1 N0 M0
Stage II	T2 N0 M0
Stage III	T3 N0 M0; T1-3 N1 M0
Stage IVA	T4a N0-1 M0; T1-4a N2 M0
Stage IVB	T4b N0-3 M0; T1-4b N3 M0
Stage IVC	T1-4b N0-3 M1

11. Differential diagnosis of midline neck swellings.

Refer Question No. 8 December 2012 (RS2).

12. Draw a detailed diagram of the lateral wall of the nose.

Refer Question No. 3 December 2007 (RS2).

■ SHORT ANSWERS

13. Tracheostomy tubes.

Refer Question No. 12 June 2016 (RS2).

14. Eagle's syndrome.

Refer Question No. 12 June 2010 (RS2).

15. Fossa of Rosenmüller.

- Also called pharyngeal recess.

Location

- At base of skull, behind ostium of Eustachian tube.

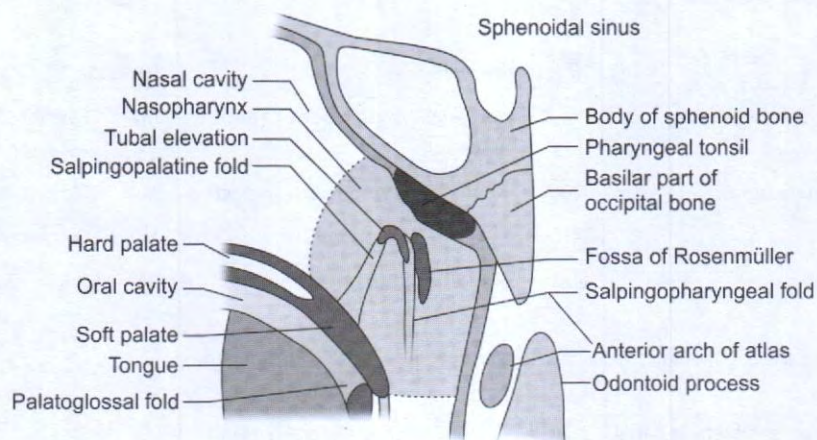


Figure 3: Fossa of Rosenmüller

Boundaries

♦ Anteriorly	Eustachian tube Levator palati muscle
♦ Posteriorly	Posterior pharyngeal wall mucosa overlying pharyngobasilar fascia
♦ Superiorly	Base of skull till foramen lacerum and floor of carotid canal
♦ Posterolateral (apex)	Carotid canal opening Petrus bone apex (posteriorly) Foramen ovale and spinosum (laterally)
♦ Laterally	Tensor palatini muscle Mandibular nerve (in prestyloid compartment)
♦ Medially	Nasopharyngeal cavity

Contents

- Retropharyngeal lymph node (Node of Rouviere).

Clinical Significance

- Common site for nasopharyngeal carcinoma.

16. MacEwen's triangle.

Refer Question No. 17 December 2013 (RS2).

17. Weber's Test.

Refer Question No. 18 December 2007 (RS2).

18. Plunging ranula.

Refer Question No. 15 June 2011 (RS2).

19. Management of acute epiglottitis.

Refer Question No. 8 June 2008 (RS2).

20. Vocal nodule.

Refer Question No. 9 June 2016 (RS2).

21. Anosmia.

- Anosmia is total loss of sense of smell.

Causes

- Nasal obstruction due to nasal polypi, enlarged turbinates
- Edema of mucous membrane—common cold, allergic or vasomotor rhinitis
- Atrophic rhinitis (degenerative disorder of nasal mucosa)
- Peripheral neuritis (toxic or influenzal) of olfactory nerves
- Injury to olfactory nerves or olfactory bulb—fractures of anterior cranial fossa
- Intracranial lesions like abscess, tumor or meningitis due to pressure on olfactory tracts
- Central lesions in the brain.

Most common causes of anosmia are sinonasal disease, post-upper respiratory tract infection and trauma (injury to olfactory nerves at cribriform plate or brain injury)

22. Saddle nose.

- Saddle nose is depression of nasal dorsum.

Etiopathogenesis

Etiology			Pathology
Traumatic	Iatrogenic	Pathological (destruction of septal cartilage)	
♦ Depressed fracture of nose (MC)	♦ Excessive removal of septum in SMR	♦ Septal hematoma ♦ Septal abscess ♦ Leprosy ♦ Tuberculosis ♦ Syphilis	♦ Involves either bony, cartilaginous or both components of nasal dorsum

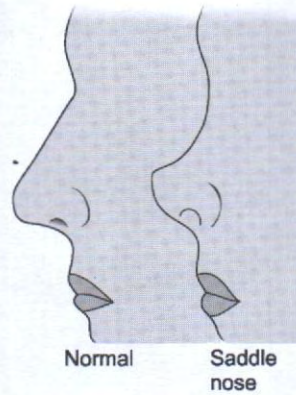


Figure 4: Saddle nose—etiopathogenesis

Treatment

Operative

- Augmentation rhinoplasty
 - Involves filling of dorsum with
 - Cartilage (from nasal septum or auricle)
 - Bone (from iliac crest)
 - Synthetic (silicone or teflon implants).

MBBS PHASE III EXAMINATION

JUNE 2015

(Revised Scheme 2 & 3)

■ LONG ESSAYS

1. Discuss the etiopathology, clinical features, management and complications of juvenile laryngeal papilloma.

Refer Question No. 11 December 2007 (RS2).

2. Discuss the etiology, clinical features and management of acute suppurative otitis media.

Refer Question No. 1 December 2012 (RS2).

■ SHORT ESSAYS

3. Waldeyer's rings.

- Waldeyer's ring is a subepithelial collection of lymphoid tissue scattered in circular fashion in nasopharynx
- At some places, this lymphoid tissue is aggregated to form masses.

Components

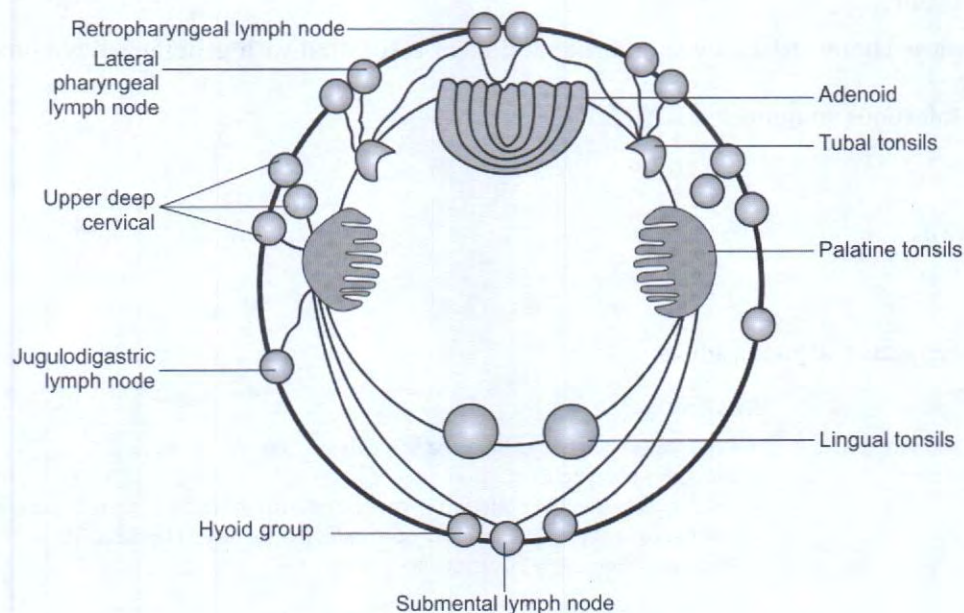


Figure 1: Waldeyer's ring—components

Components	Location
a. Palatine tonsils	♦ In palatine fossa between anterior and posterior pillars of fauces in lateral wall
b. Nasopharyngeal tonsil (adenoids)	♦ At junction of roof and posterior wall of nasopharynx in midline
c. Tubal tonsils	♦ In fossa of Rosenmuller behind opening of Eustachian tube
d. Lingual tonsil	♦ Upper surface of posterior 1/3rd of tongue, one on each side and continues with lower ends of palatine tonsils
e. Lateral pharyngeal bands	♦ Descend from tubal tonsil, behind posterior faucial pillars
f. Pharyngeal nodules	♦ In subepithelial layer of posterior pharyngeal wall

Features

- Lymphoid tissue of Waldeyer's ring exhibit efferent vessels but no afferents
- They drain into upper deep cervical lymph nodes
- Waldeyer's ring changes with age, i.e. lymphoid tissue is small at birth and increases till age of 8–10 years (physiological hypertrophy) followed by gradual receding in size to become steady by 20 years of age.

Importance (Functions)

- Act as first line of defence by way of localization of infection and development of immunity against invading organism by
 - Formation of lymphocytes and plasma cells
 - Antibody formation in early years of life
 - Protecting lower respiratory tract by guarding entry of air passage
 - Continuous monitoring of different types of invading microorganism.

4. Middle ear transformer mechanism.

Refer Question No. 1 December 2007 (RS2).

5. Glandular fever.

- Glandular fever is characterized by sore throat and fever associated with generalized lymphadenopathy and splenomegaly
- Also called as infectious mononucleosis.

Etiology

- Epstein-Barr virus.

Clinical Features

- Often affects teenagers and young adults.

Symptoms	Signs
♦ High grade fever and sore throat	♦ Petechiae at junction of hard and soft palate (characteristic feature)
♦ Marked local discomfort	♦ Exudative pharyngitis
	♦ Gross enlargement of both tonsils, congested and covered with membranous exudates (slough)
	♦ Enlargement of jugulodigastric lymph nodes (hence name glandular fever)
	♦ Hepatosplenomegaly (occasionally)

Laboratory Findings

- White blood count is normal in 1st week and rises in 2nd week
- Lymphocytosis (50% of which 10% are abnormal)—atypical lymphocytes are diagnostic
- Paul-Bunnell test shows high titres of heterophil antibody.

Treatment

- **Symptomatic** treatment
- **Antibiotics** in secondary bacterial infections (Avoid ampicillin as it causes skin rashes)
- **Management of upper airway obstruction** either tonsillectomy or tracheostomy
- **Intravenous steroids** for severe cases.

6. Tinnitus.

- Tinnitus is hearing of adventitious sound in ear that is not attributed to any external sound
- Tinnitus (Latin: tinnire = to ring).

Classification

Subjective	Objective (rare)
♦ Sounds heard only by patient	♦ Sounds heard even by examiner using stethoscope

Etiology

Otolological		Non-otological	Psychological (idiopathic)
Subjective (SOFIA PAPA Makes Onion Omelet)	Objective (GAMPA)		
<ul style="list-style-type: none"> ♦ Sensorineural hearing loss ♦ Otitis media (acute, chronic, secretory) ♦ Fluid in middle ear ♦ Impacted wax ♦ Abnormally patent Eustachian tube ♦ Presbycusis ♦ Acoustic trauma ♦ Poststapedectomy ♦ Acoustic neuroma ♦ Meniere's disease ♦ Otosclerosis ♦ Ototoxicity 	<ul style="list-style-type: none"> ♦ Glomus tumor ♦ Aneurysm of carotid, occipital, superficial temporal arteries and aortic arch ♦ Myoclonus of Tensor tympani and stapedius ♦ Palatal myoclonus ♦ AV shunts 	<ul style="list-style-type: none"> ♦ Disorders of CNS ♦ Epilepsy Arteriosclerosis ♦ Disseminated sclerosis ♦ Paget's disease ♦ Leukemia ♦ Hypoglycemia ♦ Anemia ♦ Migraine ♦ Hypertension ♦ Hypotension 	

Clinical Features

- Usually unilateral but may affect both ears.

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Sound in ear of varying pitch and loudness ♦ May be continuous or intermittent ♦ Described as familiar monotonous sounds like roaring, hissing, swishing, rustling or clicking ♦ Worse at night (due to loss of masking effect of ambient noise from environment) ♦ In conductive deafness, noise heard during eating, speaking or even respiration 	<ul style="list-style-type: none"> ♦ Tinnitus may be synchronous with pulse (vascular lesions) or respiration (abnormally patent Eustachian tube) ♦ Clicking sound (due to clonic contraction of soft palatal muscles in palatal myoclonus)

Investigations

Estimation of intensity of tinnitus	Hemogram	MRI angiography
♦ Estimated by measuring intensity of sound which masks tinnitus and plotted as tinnitogram	♦ For anemia	♦ To diagnose vascular disorders

Differential Diagnosis

- Auditory hallucinations.

Treatment

<i>Conservative</i>		<i>Operative</i>
<i>Supportive</i>	<i>Specific</i>	
<ul style="list-style-type: none"> ♦ Reassurance and psychotherapy ♦ Techniques of relaxation and biofeedback ♦ Sedation and tranquillizers in initial stages <ul style="list-style-type: none"> – Lidocaine HCl 100 mg rapid IV – Carbamazepine 200 mg TID – Tocainide HCl 200 mg TID ♦ Making of tinnitus using loudly clicking clock, fan, etc. at night and tinnitus maskers during daytime 	<ul style="list-style-type: none"> ♦ Treatment of underlying condition 	<ul style="list-style-type: none"> ♦ Stellate ganglion block ♦ Cervical sympathectomy ♦ Labryinthectomy (in very severe cases) ♦ Cochlear nerve section ♦ Prefrontal lobectomy ♦ Chorda tympani neurectomy

7. Secondary hemorrhage.

- Secondary hemorrhage is a delayed postoperative complication of tonsillectomy occurring on 5th–10th postoperative day
- Also occurs as postoperative complication of adenoidectomy.

<i>Cause</i>	<i>Precipitating factor</i>
♦ Sepsis (infection) of tonsillar fossa	♦ Premature separation of slough

Clinical Features

- Usually, heralded by bloodstained sputum or mild bleeding
- May be profuse bleeding.

Treatment

<i>Conservative</i>	<i>Operative</i>
<ul style="list-style-type: none"> ♦ Removal of clot ♦ Topical application of dilute adrenaline or hydrogen peroxide with pressure ♦ Antiseptic mouthwash ♦ Parenteral broad-spectrum antibiotic including tinidazole/metronidazole ♦ Cold liquid diet ♦ Blood/plasma transfusion depending upon blood loss 	<ul style="list-style-type: none"> ♦ Electrocauterization of bleeding vessel under GA for profuse bleeding ♦ Inter-pillar suturing in persistent bleeding ♦ External carotid ligation if bleeding is uncontrollable

8. Blood supply of nose.

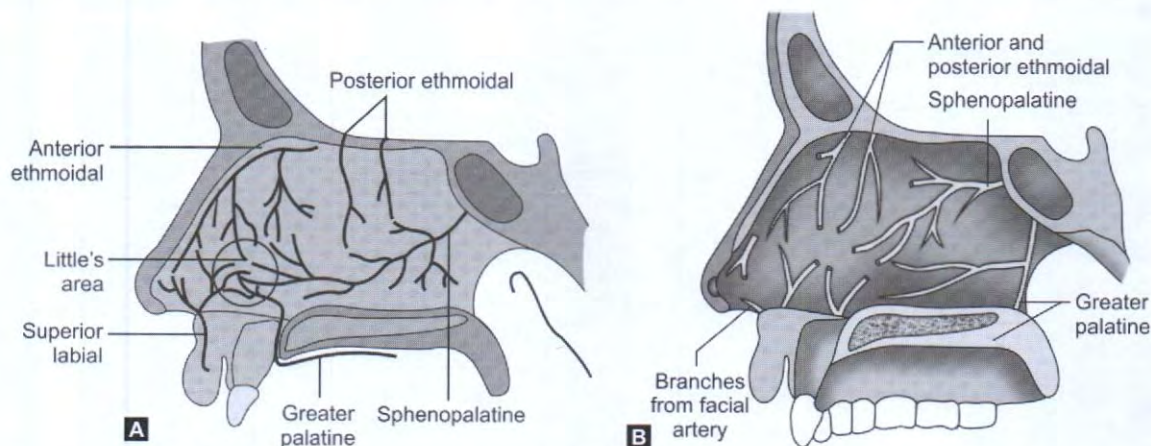
- Nose is mainly supplied by external carotid artery (area above middle turbinate) and by internal carotid artery (area below middle turbinate).

Arteries Supplying Nose

External carotid artery			Internal carotid artery	
↓	↓		↓	
Facial artery	Maxillary artery		Ophthalmic artery	
↓	↓	↓	↓	↓
Superior labial artery	Greater palatine artery	Sphenopalatine artery	Anterior ethmoidal artery	Posterior ethmoidal artery

Blood Supply of Nose

Part	Nasal septum	Lateral wall of nose
a. Anterosuperior	Anterior and posterior ethmoidal arteries	Anterior and posterior ethmoidal arteries
b. Anteroinferior	Septal branches of superior labial artery, a branch of facial artery	Branches from facial artery
c. Posterosuperior	Sphenopalatine artery (main artery)	Few branches from sphenopalatine artery
d. Posteroinferior	Branches of greater palatine artery	Branches of greater palatine artery



Figures 2A and B: (A) Blood supply of nose—nasal septum; (B) Blood supply of nose—lateral wall of nose

Significance

- Anteroinferior part or vestibule of nasal septum contains anastomoses between septal ramus of superior labial branches of facial artery, branch of sphenopalatine artery, greater palatine and of anterior ethmoidal artery which forms a larger capillary network called Kiesselbach's plexus
- This is a common site (Little's area) of epistaxis.

9. Functional endoscopic sinus surgery.

Refer Question No. 12 December 2007 (RS2).

10. Indications for tracheostomy.

Refer Question No. 9 December 2007 (RS2).

11. Juvenile nasopharyngeal angiofibroma (JNA).

Refer Question No. 1 December 2009 (RS2).

12. Management of sinonasal polyposis.

Refer Question No. 2 December 2013 (RS2).

SHORT ANSWERS**13. Weber's test.**

Refer Question No. 18 December 2007 (RS2).

14. Etiology of stridor in children.

Refer Question No. 1 June 2016 (RS2).

15. Bone-anchored hearing aids.

Refer Question No. 10 December 2011 (RS2).

16. Piriform sinus.

- Piriform sinus is a recess on either side of laryngeal orifice.
- Also called as piriform recess, piriform fossa or smuggler's fossa.

Boundaries

♦ Superiorly	♦ Pharyngoepiglottic fold
♦ Inferiorly	♦ Lower border of cricoids
♦ Medially	♦ Aryepiglottic fold ♦ Posterolateral surface of arytenoids and cricoids cartilages
♦ Laterally	♦ Thyroid cartilages ♦ Thyrohyoid membrane

Function

- Piriform fossa is involved in speech.

Significance

- Piriform fossa forms lateral channel for food and is a common site for entrapment of food or foreign body
- Piriform sinus is subsite of hypopharynx and this distinction is important for staging and treatment of head and neck cancers
- Internal laryngeal nerve runs submucosally in piriform fossa thus is easily accessible for anesthesia
- Recurrent laryngeal nerve lies deep to mucosa here and any damage to mucosa of this region may affect this nerve.

17. Laryngeal papilloma.

Refer Question No. 11 December 2007 (RS2).

18. Intratympanic muscles.

Refer Question No. 1 June 2012 (RS2).

19. Nasal furuncle.

- Furunculosis is acute infection of hair follicle.

Etiology

Causative agent	Predisposing factors	Location
♦ <i>Staphylococcus aureus</i>	♦ Trauma from picking of nose or ear, plucking nasal vibrissae ♦ Using contaminated hearing aids, stethoscope, ear mould, etc. ♦ Diabetes, immunocompromised individuals	♦ Nasal vestibule

Clinical Features

- Usually single lesion but may be multiple.

Symptoms	Signs
♦ Severe pain	♦ Tenderness ♦ Small lesion surrounded by swelling and edema ♦ Enlargement of draining lymph nodes

Treatment

<i>Conservative</i>		<i>Operative</i>
<i>Supportive</i>	<i>Specific</i>	
<ul style="list-style-type: none"> ♦ Warm compress ♦ 10% ichthamol glycerine ear pack 2–3 times daily (provides splintage and reduces pain and edema) 	<ul style="list-style-type: none"> ♦ Topical and systemic antibiotics to control infection ♦ Analgesics to relieve pain 	<ul style="list-style-type: none"> ♦ Incision and drainage if swelling turns fluctuant

Complications

- Cellulitis of upper lip, mastoid skin
- Septal abscess
- Cavernous sinus thrombosis (if squeezed or incised prematurely).

20. Plummer-Vinson syndrome.

Refer Question No. 9 December 2014 (RS2).

21. Nonhealing midline granuloma.

Refer Question No. 11 June 2009 (RS2).

22. Signs of chronic tonsillitis.

- Chronic tonsillitis usually results from inadequately treated acute tonsillitis.

Etiopathogenesis

<i>Etiology</i>	<i>Causative agent</i>	<i>Predisposing factors</i>	<i>Pathogenesis</i>
<ul style="list-style-type: none"> ♦ Recurrent acute tonsillitis ♦ Subclinical tonsillar infections 	<ul style="list-style-type: none"> ♦ β-hemolytic streptococci 	<ul style="list-style-type: none"> ♦ Chronic sinusitis ♦ Chronic dental infection 	<ul style="list-style-type: none"> ♦ Recurrent tonsillitis → minute abscesses within lymphoid follicle → abscess walled of fibrous tissue → surrounded by inflammatory cells → chronic tonsillitis

Types (Pathology)

<i>Chronic follicular tonsillitis</i>	<i>Chronic parenchymatous tonsillitis</i>	<i>Chronic fibroid tonsillitis</i>
<ul style="list-style-type: none"> ♦ Tonsillar crypts filled with infected cheesy material appearing as yellowish spots on surface ♦ Occurs usually in adults 	<ul style="list-style-type: none"> ♦ Hyperplasia of lymphoid follicles in tonsillar parenchyma leading to uniform hypertrophy of tonsils with congested and lengthened anterior pillar ♦ Occurs usually in children 	<ul style="list-style-type: none"> ♦ Small but infected tonsils

Clinical Features

- Mostly affects children and young adults.

<i>Symptoms</i>	<i>Cardinal signs</i>
<ul style="list-style-type: none"> ♦ History of recurrent sore throat or acute tonsillitis ♦ Chronic irritation in throat with cough ♦ Bad taste in mouth ♦ Foul breath due to pus in crypts ♦ Thick speech ♦ Difficulty in swallowing ♦ Choking spells in night 	<ul style="list-style-type: none"> ♦ Persistent congestion of anterior pillars (compared to rest of pharyngeal mucosa) ♦ Positive tonsillar squeeze (Ervin-Moore sign) <ul style="list-style-type: none"> – Expression of purulent cheesy material from tonsillar crypts on pressure on anterior pillar ♦ Nontender enlargement of jugulodiagastic lymph nodes bilaterally (further enlargement and tenderness during acute attack) ♦ Varying degree of tonsillar enlargement (chronic parenchymatous type) <ul style="list-style-type: none"> – Grade I Congested tonsils located within tonsillar fossa – Grade II Hypertrophied tonsils limited to tonsillar brim – Grade III Hypertrophied tonsils extend beyond pillars but not touching each other – Grade IV Tonsils meeting in midline (Kissing tonsils)

Contd...

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<i>Symptoms</i>	<i>Cardinal signs</i>
	<ul style="list-style-type: none"> ♦ Yellowish beads of pus on medial surface (chronic follicular type) ♦ Small tonsils but express frank pus or cheesy material on pressing anterior pillar (chronic fibroid type) ♦ Features of cor pulmonale (long-standing chronic parenchymatous type)

Investigations

<i>Blood investigations</i>	<i>Microbiological investigations</i>	<i>Radiography</i>	<i>Diagnostic nasal endoscopy</i>
<ul style="list-style-type: none"> ♦ Hemogram, total and differential count, ESR, platelet count, peripheral smear ♦ Bleeding time, clotting time, prothrombin time, activated prothrombin time ♦ Blood grouping 	<ul style="list-style-type: none"> ♦ ASO titer ♦ Throat swab for culture and sensitivity 	<ul style="list-style-type: none"> ♦ X-ray (lateral view of neck) to rule out coexistent adenoid hypertrophy 	<ul style="list-style-type: none"> ♦ To find out any nasal pathology

Treatment

<i>Conservative</i>	<i>Operative</i>
<ul style="list-style-type: none"> ♦ Improvement of general health ♦ Nutritious diet and vitamins ♦ Antibiotics to control infection ♦ Analgesics to relieve pain ♦ Treatment of coexistent infections in nasal and oral cavity 	<ul style="list-style-type: none"> ♦ Tonsillectomy

Complications

- Peritonsillar abscess
- Parapharyngeal abscess
- Intratonsillar abscess
- Tonsilloliths
- Tonsillar crypts
- Septic foci for rheumatic fever, acute glomerulonephritis, ocular and dermatological disorders.

MBBS PHASE III EXAMINATION

DECEMBER 2015

(Revised Scheme 2 & 3)

■ LONG ESSAYS

1. Discuss the etiopathology, clinical features and management of laryngotracheal trauma.

Etiopathology

<i>Etiology</i>	<i>Pathology</i>
<i>Blunt injuries</i> <ul style="list-style-type: none">♦ Road traffic accidents (neck striking against steering wheel or front panel)—MC♦ Blow or kick on neck♦ Neck hitting against a stretched wire or cable♦ Strangulation <i>Penetrating injuries</i> <ul style="list-style-type: none">♦ With sharp instruments or firearms <i>Others</i> <ul style="list-style-type: none">♦ Ingestion of corrosives♦ Intubation trauma	<ul style="list-style-type: none">♦ Abrasion or contusion on skin♦ Hematoma and edema of supraglottic or subglottic region♦ Tears in laryngeal or pharyngeal mucosa → subcutaneous emphysema♦ Dislocation of cricoarytenoid joints (Arytenoid cartilage may be displaced anteriorly, dislocated or avulsed)♦ Dislocation of cricothyroid joint → causes recurrent laryngeal nerve paralysis♦ Fracture (isolated or comminuted) of laryngeal framework (seen commonly adults above 40 years because of its calcification whereas rare in children due to resilient cartilages)<ul style="list-style-type: none">– Fractures of hyoid bone– Fractures of thyroid cartilage (vertical or transverse)– Fracture of upper part results in avulsion of epiglottis and one or both false cords– Fractures of lower part may displace or disrupt true vocal cords– Fractures of cricoid cartilage– Fractures of upper tracheal rings♦ Separation of trachea from cricoid cartilage and retraction into upper mediastinum → associated with injury to recurrent laryngeal nerve

Clinical Features

<i>Symptoms</i>	<i>Signs</i>
<ul style="list-style-type: none">♦ Stridor (due to laryngeal edema or hematoma)♦ Hoarseness of voice or aphonia♦ Painful and difficult swallowing; accompanied by aspiration of food, blood and secretion♦ Local pain in larynx (more marked on speaking or swallowing)♦ Hemoptysis (from bleeding due to tears in laryngeal or tracheal mucosa)	<ul style="list-style-type: none">♦ Bruises or abrasions of skin♦ Tenderness of laryngeal area♦ Subcutaneous emphysema which increases on coughing (due to mucosal tears)♦ Flattening of thyroid prominence and contour of anterior cervical region with inability to palpate thyroid notch (loss of normal contour of neck)♦ Fracture displacements of thyroid or cricoid cartilage or hyoid bone♦ Gap and bony crepitus between fractured fragments of hyoid bone, thyroid or cricoid cartilages may be felt separation of cricoid cartilage from larynx or trachea♦ There may be other injuries to head, cervical spine, chest, abdomen and extremities

Investigations

a. Indirect laryngoscopy (most valuable investigation)	<ul style="list-style-type: none">♦ Reveals location and degree of edema, hematoma, mucosal lacerations, posterior displacement of epiglottis, exposed fragments of cartilage, asymmetry of glottis or laryngeal inlet
b. Direct laryngoscopy (not routinely done)	<ul style="list-style-type: none">♦ Rarely informative in early period following injury and if performed, may precipitate respiratory distress and necessitate immediate tracheostomy♦ If required, fiberoptic laryngoscopy is preferred

Contd...

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c. Radiology	
i. X-rays	<ul style="list-style-type: none"> ♦ Soft tissue lateral X-ray of neck reveals subcutaneous emphysema, swelling of laryngeal mucosa, displacement of epiglottis, fracture displacements of hyoid bone, thyroid and cricoid cartilages or change in configuration of air column ♦ X-ray chest for pneumothorax and gastrograffin swallow for esophageal tears
ii. 3D CT scan	<ul style="list-style-type: none"> ♦ Very valuable in assessing moderately severe or severe injuries of larynx

Treatment

Conservative	Operative
<ul style="list-style-type: none"> ♦ Hospitalize and observe for respiratory distress ♦ Voice rest ♦ Humidification of inspired air ♦ Full dose steroid therapy started immediately (Helps to resolve edema and hematoma and prevent scarring and stenosis) ♦ Antibiotics (to prevent perichondritis and cartilage necrosis) 	<p>a. Tracheostomy</p> <ul style="list-style-type: none"> – Tracheostomy is preferred as endotracheal intubation in these cases may be difficult and hazardous <p>b. Open reduction</p> <p><i>Timing</i></p> <ul style="list-style-type: none"> – 3–5 days after injury and not delayed beyond 10 days <p><i>Techniques</i></p> <ul style="list-style-type: none"> – Fractures of hyoid bone, thyroid or cricoid cartilage are wired and replaced in their anatomic positions and immobilized using titanium miniplates – Mucosal lacerations repaired with catgut and any loose fragments of cartilage removed – Epiglottis is anchored in its normal position and if already avulsed then excised – Arytenoid cartilages can be repositioned in their normal position or may be removed if completely avulsed – In laryngotracheal separation, end-to-end anastomosis is done – Internal splintage of laryngeal structures is done with a laryngeal stent, or silicone tube and left in-situ for 2–6 weeks – Webbing of anterior commissure can be prevented by a silastic keel

Complications

- Laryngeal stenosis (supraglottic, glottic or subglottic)
- Perichondritis
- Laryngeal abscess
- Vocal cord paralysis.

2. Discuss the etiopathology, clinical features, complications and management of acute mastoiditis.

Refer Question No. 3 December 2009 (RS2).

SHORT ESSAYS**3. Orbital complications of sinusitis.**

- Sinusitis is known to cause orbital complications due to close relation between ethmoid, frontal and maxillary sinus with orbit and its contents.

Etiopathogenesis

Etiology	Spread of infection
<ul style="list-style-type: none"> ♦ Ethmoidal sinusitis (more common as they are separated from orbit by a lamina papyracea, a thin lamina of bone) ♦ Frontal sinusitis ♦ Maxillary sinusitis 	<ul style="list-style-type: none"> ♦ Through thin lamina papyracea, osteitis or as thrombophlebitis of ethmoidal veins

**Orbital complications of sinusitis—
“OOOPSS”**

- Orbital cellulitis
- Orbital abscess
- Orbital apex syndrome
- Preseptal inflammatory edema of lids
- Subperiosteal abscess
- Superior orbital fissure syndrome

Orbital Complications of Sinusitis

- a. Inflammatory edema of lids (only reactionary)—preseptal cellulitis
 - First indication of orbital involvement
 - Involves only preseptal space (lies in front of orbital septum)
 - Upper lid is swollen in frontal, lower lid in maxillary and both upper and lower lids in ethmoidal sinusitis
 - No erythema or tenderness of the lids which characterizes lid abscess
 - Eyeball movements and vision are normal.
- b. Subperiosteal abscess (collection of pus outside the bone under periosteum)

<i>Sinusitis</i>	<i>Location of abscess</i>	<i>Displacement of eyeball</i>
i. Ethmoidal	On medial wall of orbit	Forward, downward and laterally
ii. Frontal	Just above and behind medial canthus	Downwards and laterally
iii. Maxillary	In floor of orbit	Upwards and forwards

- c. Orbital cellulitis

Etiopathogenesis

- Pus from subperiosteal abscess breaks through periosteum and finds its way into orbit, spreading between orbital fat, extraocular muscles, vessels and nerves.

Clinical features

<i>Symptoms</i>	<i>Signs</i>
<ul style="list-style-type: none"> ♦ Partial or total visual loss (sometimes permanent) ♦ High fever 	<ul style="list-style-type: none"> ♦ Edema of lids ♦ Chemosis of conjunctiva ♦ Exophthalmos ♦ Impairment of ocular motility

Complications

- Meningitis
 - Cavernous sinus thrombosis.
- d. Orbital abscess
 - Usually forms along lamina papyracea or floor of frontal sinus
 - Clinical features similar to orbital cellulitis
 - Diagnosed by CT scan or USG of orbit
 - Treated with IV antibiotics and drainage of abscess and affected sinus (ethmoidectomy or trephination of frontal sinus).
 - e. Superior orbital fissure syndrome
 - Rarely seen as complication of sphenoid sinusitis
 - Manifests as deep orbital pain, frontal headache and progressive paralysis of CN VI, III and IV (in that order).
 - f. Orbital apex syndrome
 - Superior orbital fissure syndrome with additional involvement of optic nerve and maxillary division of trigeminal (V2).

Investigations

- Radiography
 - CT scan and ultrasound will confirm abscess formation.

Treatment

<i>Conservative</i>	<i>Operative</i>
a. Antibiotics <ul style="list-style-type: none"> – Indicated in orbital inflammation and cellulitis – Given either orally or intravenous based on severity – Usually indicated are 3rd generation cephalosporin and metronidazole or clindamycin to cover anaerobic organisms and a penicillin or vancomycin to cover gram-positive organisms (in orbital cellulitis) b. Anti-inflammatory drugs c. Topical nasal vasoconstrictors	a. Surgical drainage <ul style="list-style-type: none"> – Drainage of sinus or abscess to prevent permanent orbital sequel and intracranial complications – Is done with (or without) endoscopic ethmoidectomy, antrostomy or frontal sinus ostium widening

4. Ludwig's angina.

Refer Question No. 5 December 2011 (RS2).

5. Referred otalgia.

Refer Question No. 3 June 2010 (RS2).

6. Sudden sensorineural hearing loss.

- Sudden hearing loss is sensorineural hearing loss developed over a period of hours or within 3 days.

Etiology (In The Very Inner Ear Too, No Major Pathology)

a. Idiopathic (MC)	
b. Trauma	<ul style="list-style-type: none"> ♦ Head injury with fracture of petrous part of temporal bone ♦ Iatrogenic (Ear operations) ♦ Barotrauma ♦ Noise induced ♦ Spontaneous rupture of cochlear membrane
c. Vascular	<ul style="list-style-type: none"> ♦ Hemorrhage in labyrinth (trauma, hypertension, leukemia, purpura, etc.) ♦ Vascular pathology of labyrinthine or cochlear artery (embolism, thrombosis or vasospasm)
d. Infections	<ul style="list-style-type: none"> ♦ Mumps ♦ Herpes zoster ♦ Meningitis ♦ Encephalitis ♦ Syphilis ♦ Otitis media
e. Ear	<ul style="list-style-type: none"> ♦ Meniere's disease ♦ Cogan's syndrome ♦ Large vestibular aqueduct
f. Toxic	<ul style="list-style-type: none"> ♦ Ototoxic drugs ♦ Insecticides
g. Neoplastic	<ul style="list-style-type: none"> ♦ Acoustic neuroma ♦ Carcinomatous neuropathy ♦ Metastasis in cerebellopontine (CP) angle
h. Miscellaneous	<ul style="list-style-type: none"> ♦ Multiple sclerosis ♦ Hypothyroidism ♦ Sarcoidosis ♦ Raised intracranial pressure
i. Psychogenic	

Clinical Features

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Sudden hearing loss within period of hours or few days ♦ Loss may be partial or complete ♦ Mostly unilateral ♦ Accompanied by tinnitus or temporary spell of vertigo 	<ul style="list-style-type: none"> ♦ Deterioration of >35 dB in at least 3 adjacent frequencies within 3 days

Investigations

Audiometry	Vestibular tests	Laboratory tests	Radiology
<ul style="list-style-type: none"> ♦ Deterioration of >35dB in atleast 3 adjacent frequencies 		<ul style="list-style-type: none"> ♦ For syphilis, diabetes, hypothyroidism, blood disorders and lipid profile 	<ul style="list-style-type: none"> ♦ X-ray, CT and MRI of temporal bone

Treatment

Supportive	
a. Bed rest	♦ In head raised position
b. Sedatives	♦ To relieve anxiety and associated giddiness
c. Steroid therapy	<ul style="list-style-type: none"> ♦ Prednisolone 15 mg TID dose for 10 days followed by tailing off ♦ Helps by relieving edema
d. Inhalation of carbogen (95% O ₂ + 5% CO ₂)	♦ Improves oxygenation by increasing cochlear blood flow
e. Hyperbaric oxygen therapy	♦ Beneficial if given within 1st month of onset
f. Low molecular weight dextran	♦ Decreases viscosity of blood
g. Vasodilator drugs	<ul style="list-style-type: none"> ♦ Histamin IV in first 3 days ♦ Nylidrine hydrochloride, nicotinic acid orally or parenterally
h. Stellate ganglion block	♦ To relieve vasospasm by correcting autonomic balance
Specific	
a. Treatment of underlying cause	

Prognosis

- Spontaneous recovery in 50% patients within 15 days
- Poor prognosis after 1 month.

7. Nerve supply of larynx.

- Larynx is supplied by vagus through its branches—recurrent laryngeal nerve and internal and external branches of superior laryngeal nerve.

	Recurrent laryngeal nerve	Superior laryngeal nerve
Origin and course	<p>Right recurrent laryngeal nerve arises from vagus at level of subclavian artery, hooks around it and then ascends between trachea and esophagus</p> <p>Left recurrent laryngeal nerve arises from vagus in mediastinum at level of arch of aorta, loops around it and then ascends into neck in tracheo-esophageal groove (it has a much longer course making it more prone to paralysis)</p>	Arises from inferior ganglion of vagus, descends behind internal carotid artery and, at level of greater cornua of hyoid bone, divides into external and internal branches
Sensory supply	Below vocal cords	Above vocal cords (internal laryngeal nerve after piercing thyrohyoid membrane)
Motor supply	All muscles of larynx (except cricothyroid)	Cricothyroid

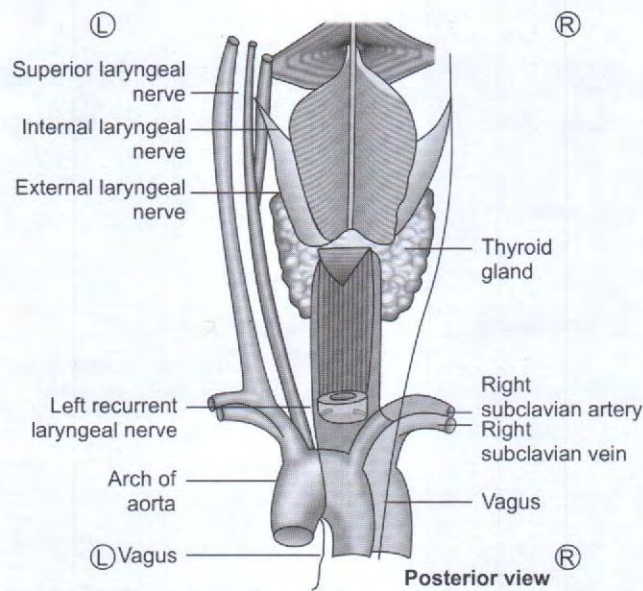


Figure 1: Course of recurrent laryngeal nerve

Revised Scheme 2 & 3

8. Septal abscess.

- Septal abscess is collection of pus within nasal septum.

Etiology

Etiology	Predisposing factors
<ul style="list-style-type: none">♦ Secondary infection of septal hematoma♦ Furuncle of nose or upper lip♦ Following typhoid or measles in immunocompromised individuals	<ul style="list-style-type: none">♦ Diabetes♦ Immunocompromised individuals

Clinical Features

Symptoms	Signs
<ul style="list-style-type: none">♦ Severe bilateral nasal obstruction♦ Pain over bridge of nose♦ Fever with chills and rigor♦ Frontal headache	<ul style="list-style-type: none">♦ Red and swollen skin over nose♦ Tenderness over bridge of nose♦ Smooth bilateral fluctuating swelling of septum with pus pointing♦ Purulent discharge (if abscess ruptured)♦ Congested septal mucosa♦ Enlarged and tender submandibular lymph nodes

Treatment

Conservative	Operative
<ul style="list-style-type: none">♦ Systemic antibiotics for 10days	<ul style="list-style-type: none">a. Wide bore needle aspiration<ul style="list-style-type: none">– Aspiration of pus at early stageb. Incision and drainage<ul style="list-style-type: none">– Incision on most dependent part of abscess cavity and drainage of pus and necrosed pieces of cartilage using suction– Need to be repeated daily for 2–3 days

Complications

- Necrosis of septal cartilage
- Depression of dorsum in supratip area (due to necrosis)
- Septal perforation
- Spread of infection leading to meningitis, cavernous sinus thrombosis, facial cellulitis.

9. Middle ear cleft.

Refer Question No. 1 Oct. 2012 (RS2).

10. Physiology of nose and paranasal sinuses.

- Nose and paranasal (PNS) air sinuses are important components of upper respiratory passage and play important role in respiration.

Functions of Nose and Paranasal Air Sinuses

- Natural pathway for breathing
 - Nose is natural pathway for breathing
 - During quiet respiration, inspiratory air current passes through middle part of nose between turbinates and nasal septum
 - During expiration, air current follows same course as during inspiration, but entire air current is not expelled directly through the nares.

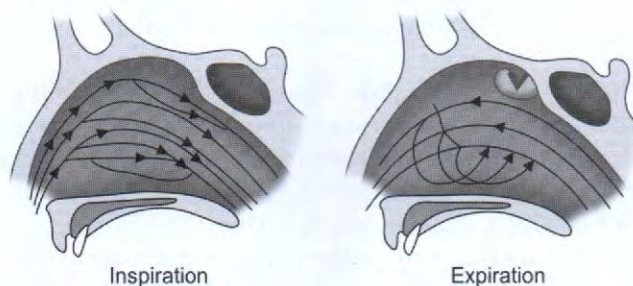


Figure 2: Flow of air current during respiration

- Friction offered at limen nasi converts it into eddies under cover of inferior and middle turbinates and this ventilates paranasal sinuses through their ostia
 - Nasal mucosa undergoes rhythmic cyclical congestion and decongestion (Nasal cycle), thus controlling airflow through nasal chambers
 - When one nasal chamber is working, total nasal respiration, equal to that of both nasal chambers, is carried out by it.
- Filtration of inhaled air
 - Nose prevents particles greater than 10 mm like dust, pollen and bacterial from reaching lungs by filtering them with hairs present in nasal cavity
 - Smaller particles of size 0.5–3 mm which are trapped by mucus secreted by nasal mucous membrane.
 - Air-conditioning and humidification of inhaled air
 - Nose along with nasal sinuses humidify and cool or warm inspired air so that inspired air attains body temperature by time it reaches lungs
 - This function is achieved by highly vascular mucosa in regions of middle and inferior turbinate and surrounding nasal septum which acts as radiator
 - Air at freezing temperature is brought to body temperature in 1/4th of second
 - Humidification is achieved by water provided by nasal mucous membrane rich in mucous and serous secreting glands.
 - Olfaction
 - Nasal cavity is lined by olfactory epithelium in its upper part which is involved in special sense of olfaction
 - Olfaction is important for pleasure and enjoyment of food and other substances.

e. Mucociliary mechanism

- Mucociliary mechanism is movement of mucous facilitated by action of cilia
- Nasal secretion is spread over normal mucosa as a continuous sheet called mucous blanket consisting of superficial mucus layer and deeper serous layer
- This blanket is floating on top of cilia which are constantly beating to carry it like a conveyer belt towards nasopharynx at speed of 5–10 mm per minute
- Cilia movement occur in two phase, i.e. rapid effective stroke wherein they reach mucous layer and slow recovery stroke wherein they bend and travel slowly in reverse direction in thin serous layer thus moving mucous blanket in only one direction
- This mucous blanket traps inspired bacteria, viruses and dust which are carried to nasopharynx to be swallowed
- Thus mucociliary mechanism is an important protective mechanism of nose.

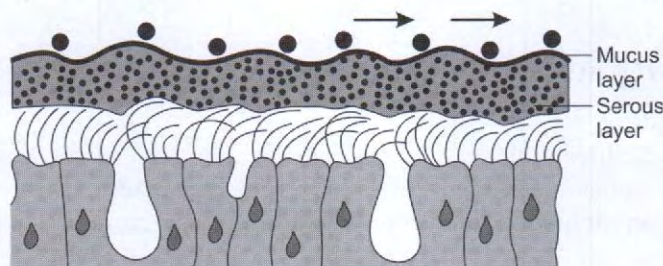


Figure 3: Mucociliary mechanism of nose

f. Protection

- Nasal secretions contain enzymes like muramidase (lysosomes), immunoglobulins (IgA and IgE) and interferons that kill microorganisms and provide immunity against upper respiratory tract infections
- Sneezing is a protective reflex which expels irritant foreign substances followed by copious nasal secretions which wash them out
- Paranasal air sinuses offer protection to brain during facial trauma
- Paranasal sinuses act as thermal insulators to protect delicate structures in orbit and cranium from intranasal temperature variations.

g. Vocalization

- Nose forms a resonating chamber for certain consonants called nasal consonants (M/N/NG)
- Paranasal sinuses offer resonance to voice.

h. Miscellaneous

- Paranasal sinuses lighten skull, thus enabling upright posture in humans.

11. Management of atrophic rhinitis.

Refer Question No. 1 December 2014 (RS2).

12. Quinsy.

Refer Question No. 7 June 2011 (RS2).

■ SHORT ANSWERS**13. Laryngomalacia.**

Refer Question No. 7 December 2009 (RS2).

14. Constrictions of esophagus.

- Esophagus is a 25 cm long fibromuscular tube, extending from lower end of pharynx (C6) to cardiac end of stomach (T11)

- It runs vertically but inclines to left from its origin to thoracic inlet and again from T7 to esophageal opening in diaphragm
- During its course it shows 3 normal constrictions (4 according to Anatomy textbooks).

Constrictions of Esophagus

Location	Vertebral level	Distance from upper incisors
a. At pharyngoesophageal junction	C6	15 cm
b. At crossing of arch of aorta and left main bronchus	T4	25 cm
c. At crossing of left main bronchus	T5	27 cm
d. Where it pierces diaphragm	T10	40 cm

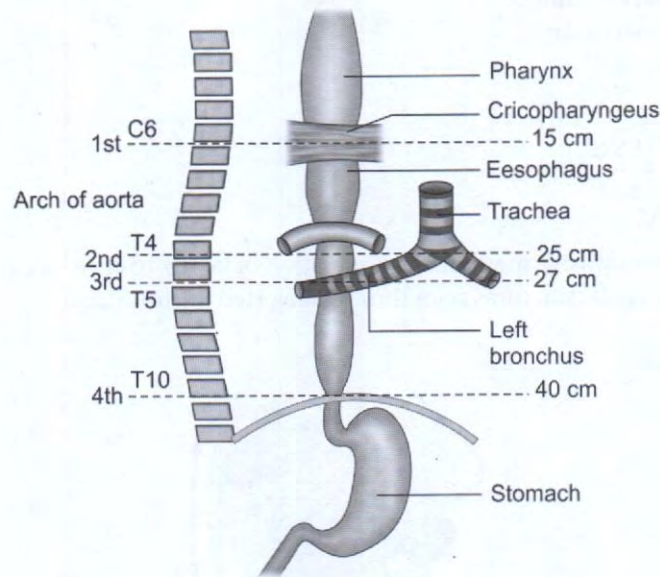


Figure 4: Constrictions of esophagus

Significance

- It is important to know the location (distance from upper incisors) for esophagoscopy
- Foreign bodies in esophagus usually are held up at these constrictions.

15. Pyriform fossa.

Refer Question No. 6 June 2009 (RS2).

16. Keratosis pharyngitis.

- Benign self-limiting condition
- Also called tonsillar keratosis.

Etiopathogenesis

Etiology	Pathology
♦ Idiopathic	♦ Hypertrophy and keratinization of overlying epithelium

Clinical Features

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Irritation and mild discomfort in throat ♦ Bad taste in mouth ♦ Feeling of foreign body 	<ul style="list-style-type: none"> ♦ Horny outgrowth on surface of tonsil, pharyngeal wall, lingual tonsil or base of tongue appearing as white or yellowish dots ♦ Firmly adherent lesions which cannot be easily removed ♦ No accompanying inflammation

Differential Diagnosis

- Acute follicular tonsillitis.

Treatment

- Spontaneously regresses within few months
- No specific treatment except reassurance
- Mandl's paint and gargles if necessary.

17. Fistula test.

Refer Question No. 3 June 2009 (RS2).

18. Indirect laryngoscopy.

- Indirect laryngoscopy is a noninvasive method of examination of larynx using a laryngeal mirror
- Called indirect because laryngeal structures seen through inverted mirror image.

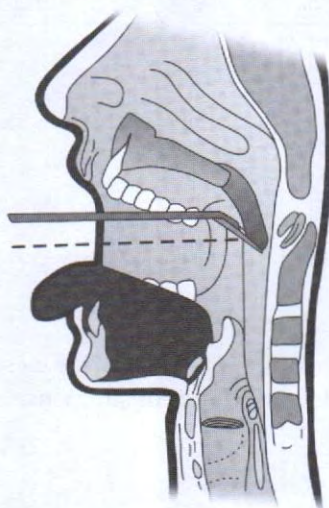


Figure 5: Indirect laryngoscopy—principle

Indications		Contraindications
Diagnostic	Therapeutic	
<ul style="list-style-type: none"> ♦ As part of routine ENT examination for diagnosis and evaluation of laryngeal diseases ♦ To take biopsy ♦ To anesthetize larynx and trachea before direct laryngoscopy or bronchoscopy 	<ul style="list-style-type: none"> ♦ Removal of small benign lesions ♦ Cauterization of ulcers 	<ul style="list-style-type: none"> ♦ Trismus ♦ Short neck

Procedure

- Ask patient to sit erect with head and chest slightly leaning towards examiner seated directly opposite
- Wrap protruded tongue of patient in gauze and hold between thumb and middle finger while keeping upper lip and moustache out of way with index finger
- Warm laryngeal mirror and test it on back of hand before introducing it in mouth (to prevent fogging of its surface caused by water vapor in patient's breath)
- Hold laryngeal mirror in right hand like a pen and introduce it through angle of mouth
- In mouth, hold it firmly against uvula and soft palate with mirror surface down without touching posterior pharyngeal wall
- Ask patient to breathe normally and quietly
- Focus light on laryngeal mirror and tilt mirror to observe structures in larynx and pharynx
- Observe and compare movements of vocal cords by asking patient to take deep inspiration (abduction of cords) and saying words like Aa (adduction of cords) and Eee (adduction and tension of cords).

Structures Seen on Indirect Laryngoscopy (Above Downwards)

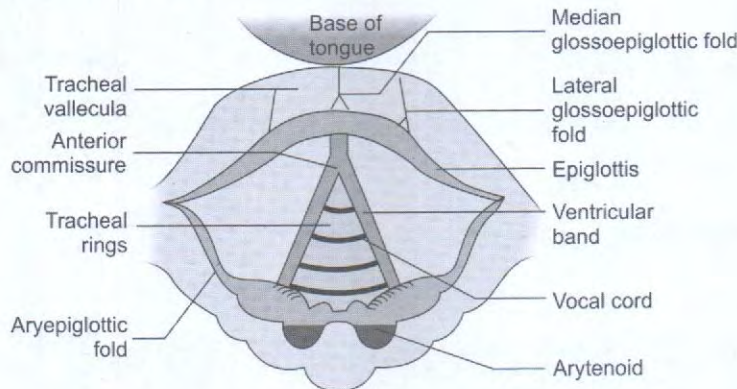


Figure 6: Indirect laryngoscopy—structures seen

Oropharynx	Larynx	Laryngopharynx
<ul style="list-style-type: none"> ♦ Posterior 1/3rd of tongue ♦ Lingual tonsils ♦ Valleculae ♦ Lateral and median glossoepiglottic fold 	<ul style="list-style-type: none"> ♦ Epiglottis ♦ Aryepiglottic fold ♦ Arytenoids, cuneiform and corniculate cartilages ♦ Pharyngoepiglottic folds ♦ Vestibule ♦ False vocal cords ♦ True vocal cords ♦ Interarytenoid area ♦ Rima glottidis ♦ Tracheal rings 	<ul style="list-style-type: none"> ♦ Posterior pharyngeal wall ♦ Pyriform fossa ♦ Postcricoid region

Advantages	Disadvantages
<ul style="list-style-type: none"> ♦ Noninvasive procedure ♦ Provides better observation of movements of vocal cords ♦ OPD procedure ♦ Does not require anesthesia 	<ul style="list-style-type: none"> ♦ Requires greater patient cooperation ♦ Provides inverted mirror image ♦ True and false vocal cords observed in one plane ♦ Difficult to view endolarynx while phonation ♦ Difficult in children

19. Thyroglossal cyst.

Refer Question No. 5 June 2012 (RS2).

20. Benign positional vertigo.

- Benign paroxysmal positional vertigo is a peripheral vestibular disorder characterized by vertigo when head is placed in certain critical position.

Etiopathogenesis

Etiology	Predisposing factors	Pathogenesis
<ul style="list-style-type: none"> Idiopathic—in majority Disorder of posterior semicircular canal 	<ul style="list-style-type: none"> Head trauma Ear infection Ear operation 	<p>Degeneration of macula of utricle</p> <p>↓</p> <p>Release of otoconical debris (consisting of crystals of calcium carbonate)</p> <p>↓</p> <p>Debris floats freely in endolymph</p> <p>↓</p> <p>Critical head position</p> <p>↓</p> <p>Settling of otoconia on cupula of posterior semicircular canal</p> <p>↓</p> <p>Displacement of cupula</p> <p>↓</p> <p>Vertigo</p>

Clinical Features

- Usually occurs between ages of 30–50 years
- More common in women
- Sudden onset.

Symptoms	Signs
<ul style="list-style-type: none"> Severe vertigo in certain head position lasting less than a minute Associated with nausea and vomiting Absence of deafness and tinnitus No other neurologic symptoms 	<ul style="list-style-type: none"> Nystagmus lasting 40 second

Investigations

Hearing tests	Caloric test and electronystagmography	Hallpike test (positional test)	X-ray spine
<ul style="list-style-type: none"> Normal 	<ul style="list-style-type: none"> May be normal 	<ul style="list-style-type: none"> Positive Fatiguable vertigo on assuming same position repeatedly (due to dispersal of otoconia) 	<ul style="list-style-type: none"> Cervical spondylosis

Treatment

Conservative

- Reassurance and antivertigo drugs.
- Epley's maneuver.

Principle

- Repositioning of otoconical debris from posterior semicircular canal back into utricle.

Position

- Patient sitting on a table such that head extends beyond edge of table when supine with face turned 45° to affected side
- Doctor standing behind patient and assistant on side.

Procedure (maneuver)

- Position 1 Make patient to lie down in head hanging position with head turned 45° Wait till vertigo and nystagmus subsides
- Position 2 Now turn head so that affected ear is up
- Position 3 Then rotate whole body and head away from affected ear to a recumbent position with face down
- Position 4 Bring back patient to sitting position with head still turned to unaffected side by 45°
- Position 5 Head turned forward and chin brought down 20°

Follow up

- Maintain upright posture for 48 hours after maneuver.

Operative

- Labyrinthectomy
- Posterior canal occlusion
- Singular neurectomy
- Vestibular nerve section.

21. Causes of trismus.

- Trismus is inability to open mouth adequately.

Etiology

<i>Acute trismus</i>		<i>Chronic trismus</i>
<i>Painless</i>	<i>Painful</i>	
<ul style="list-style-type: none"> ♦ Tetanus ♦ Tetany ♦ Strychnine poisoning 	<ul style="list-style-type: none"> ♦ Paratonsillar abscess ♦ Alveolar abscess ♦ Acute parotitis ♦ Mumps ♦ Acute temperomandibular arthritis ♦ Acute otitis externa ♦ Impacted wisdom teeth 	<ul style="list-style-type: none"> ♦ Submucous fibrosis ♦ Ankylosis of TM joint ♦ Malignancy of cheek, tonsil, palate, maxillary sinus and parotid ♦ Burns ♦ Radiation therapy

Treatment**Operative**

- a. True ankylosis
 - Excision of condyle of mandible.
- b. False ankylosis (external scarring)
 - Forming a false joint at angle of mandible by excising wedge shaped bone piece with narrow end towards alveolus in region of angle of mandible (Esmaich's operation).

22. Paracusis Willisii.

- Paracusis Willisii is ability to hear better in noisy surrounding than quite environment
- It was described originally by Willis in 1672.

Etiopathogenesis

<i>Seen in</i>	<i>Pathogenesis (Lombard, 1911)</i>
<ul style="list-style-type: none"> ♦ Otosclerosis (70%) ♦ Conductive deafness 	<ul style="list-style-type: none"> ♦ Patient can hear better in noisy surrounding because of: <ul style="list-style-type: none"> – Low pitched background noise – Low tone deafness in the patient – Unconscious raised voice by the speaker against background noise above noise level and above threshold of otosclerotic patient

MBBS PHASE III EXAMINATION

JUNE 2016

(Revised Scheme 2 & 3)

LONG ESSAYS

1. Define stridor. Enumerate the causes of stridor. How do you manage a patient of stridor?

- Stridor is abnormal, harsh, high pitched, turbulent musical breathing sound produced by turbulent airflow through narrowed air passage.

Types

Inspiratory (croup)	Expiratory (wheeze)	Biphasic
<ul style="list-style-type: none"> Seen in obstructive lesions of supraglottis or pharynx Characterized by low pitched flutter and normal voice 	<ul style="list-style-type: none"> Seen in lesions of thoracic trachea, primary and secondary bronchi Characterized by brassy barking cough and normal voice 	<ul style="list-style-type: none"> Seen in lesions of glottis, subglottis and cervical trachea Characterized by phonatory stridor and dysphonia

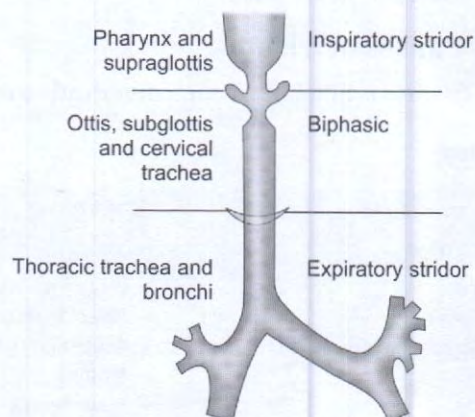


Figure 1: Stridor—types

Etiology

Congenital	Infants and children	Adults
	Acquired	
a. Nose <ul style="list-style-type: none"> Choanal atresia b. Tongue <ul style="list-style-type: none"> Macroglossia c. Mandible <ul style="list-style-type: none"> Micrognathia Pierre-Robin syndrome d. Pharynx <ul style="list-style-type: none"> Congenital dermoid e. Larynx <ul style="list-style-type: none"> Supraglottis <ul style="list-style-type: none"> Laryngomalacia Cysts Glottic <ul style="list-style-type: none"> Web 	a. Inflammatory <ul style="list-style-type: none"> Larynx <ul style="list-style-type: none"> Epiglottitis Laryngotracheitis Diphtheria Tracheobronchial <ul style="list-style-type: none"> Tracheobronchitis Outside respiratory system <ul style="list-style-type: none"> Ludwig's angina Retropharyngeal and retroesophageal abscess b. Neoplastic <ul style="list-style-type: none"> Hemangioma Lymphangioma Thymoma Juvenile multiple papillomas 	a. Trauma <ul style="list-style-type: none"> Laryngotracheal trauma and stenosis due to RTA/foreign bodies b. Tumors <ul style="list-style-type: none"> Carcinoma larynx Carcinoma pharynx Neck masses c. Infection <ul style="list-style-type: none"> Tuberculosis Neck space infections d. Allergy <ul style="list-style-type: none"> Angioneurotic edema e. Neurological <ul style="list-style-type: none"> Bilateral adductor palsy following thyroidectomy or cardiothoracic surgery

Contd...

Contd...

<i>Infants and children</i>		<i>Adults</i>
<i>Congenital</i>	<i>Acquired</i>	
<ul style="list-style-type: none"> – Cord paralysis – Cysts ♦ Subglottic – Stenosis 	<ul style="list-style-type: none"> c. Traumatic ♦ Laryngeal Injuries like burns, inhalation of fumes or swallowing of corrosives ♦ Foreign bodies ♦ Edema following endoscopy/ intubation ♦ Tracheal stenosis following intubation/ tracheostomy 	
f. Tracheobronchial <ul style="list-style-type: none"> ♦ Atresia ♦ Stenosis ♦ Tracheomalacia 	d. Miscellaneous <ul style="list-style-type: none"> ♦ Dermoid of base of tongue ♦ Lingual thyroid ♦ Laryngeal paralysis due to acquired lesions ♦ Tetanus ♦ Tetany ♦ Laryngismus stridulus 	
g. Outside respiratory system <ul style="list-style-type: none"> ♦ Vascular rings ♦ Esophageal atresia ♦ Tracheoesophageal fistula ♦ Congenital goiter ♦ Cystic hygroma 		

Clinical Features

- Stridor is physical sign of underlying disease

<i>Symptoms</i>	<i>Signs</i>
<ul style="list-style-type: none"> ♦ Age of onset determines congenital or acquired etiology ♦ May be sudden onset or gradually progressive ♦ Of short duration or long ♦ May be related to feeding ♦ Associated with cyanotic spells ♦ History of aspiration or ingestion of foreign body or laryngeal trauma 	<ul style="list-style-type: none"> ♦ Respiratory distress evident by recession in suprasternal notch, sternum, intercostals spaces and epigastrium ♦ Type and characteristics of stridor indicate location of cause ♦ Inspiratory (supraglottis or pharynx), expiratory (thoracic trachea, primary and secondary bronchi) or biphasic (glottis, subglottis and cervical trachea) ♦ Snoring or snorting sound (nasal or nasopharyngeal cause), gurgling sound and muffled voice (pharyngeal cause), hoarse cry (vocal cord), expiratory wheeze (bronchial obstruction) ♦ Associated fever indicates inflammatory cause ♦ Disappearance of stridor in prone position (laryngomalacia, micrognathia, macroglossia and innominate artery compression)

Investigations

<i>Radiography</i>	<i>Direct laryngoscopy w/o anesthesia</i>	<i>Bronchoscopy, laryngoscopy and esophagoscopy under GA</i>
<ul style="list-style-type: none"> ♦ X-ray neck (AP and lateral view) for soft tissue mass in larynx/ hypopharynx, airway patency and prevertebral widening ♦ X-ray chest (PA and lateral view) for pulmonary status, mediastinal widening, secondaries ♦ Fluoroscopy to see chest movements ♦ CT chest to detect mediastinal mass ♦ Esophagogram for atresia, fistula or aberrant vessels ♦ Angiography ♦ Xeroradiography for soft tissue lesions in neck 	<ul style="list-style-type: none"> ♦ To visualize pharynx and larynx and assess possibility of intubation 	

Treatment

<i>Supportive</i>	<i>Specific</i>
<ul style="list-style-type: none"> ♦ Hospitalization ♦ Intubation or tracheostomy or cricothyroidotomy to relieve airway obstruction in severe stridor ♦ Oxygen and humidification ♦ Antibiotics and steroids in presence of infection 	<ul style="list-style-type: none"> ♦ Treatment of underlying cause

2. Discuss the etiology, clinical features, diagnosis and management of acute suppurative otitis media.

Refer Question No. 1 April 2013 (RS2).

■ SHORT ESSAYS

3. Differences between antrochoanal and ethmoidal polyp.

Points of difference	Antrochoanal polyp	Ethmoidal polyp
♦ Age of occurrence	♦ Children and young adults	♦ Adults
♦ Etiology	♦ Infection	♦ Allergy or multifactorial
♦ Number	♦ Solitary	♦ Multiple
♦ Laterality	♦ Unilateral	♦ Bilateral
♦ Origin	♦ Maxillary sinus near ostium	♦ Ethmoidal sinuses, uncinat process, middle turbinate and middle meatus
♦ Extension	♦ Posteriorly towards choana	♦ Anteriorly
♦ Presentation	♦ Behind soft palate	♦ At nares
♦ Size and shape	♦ Trilobed with antral, nasal and choanal parts	♦ Small and grape-like masses
♦ X-ray	♦ Hazy maxillary sinus	♦ Hazy ethmoidal sinus
♦ Treatment	♦ Polypectomy either endoscopic or by Caldwell-Luc operation	♦ Polypectomy either endoscopic or by ethmoidectomy
♦ Recurrence	♦ Uncommon	♦ Common

4. Endoscopic sinus surgery.

Refer Question No. 12 December 2007 (RS2).

5. Enumerate causes of white patches in tonsil.

Refer Question No. 12 December 2011 (RS2).

6. Chronic retropharyngeal abscess.

Refer Question No. 2 December 2009 (RS2).

7. Etiology, clinical features and management of adenoiditis.

- Chronic inflammation or enlargement of adenoids is called adenoiditis.
- Also called adenoid hypertrophy or adenoids.

Etiology		Predisposing factors
Physiological	Pathological (Causative organisms)	
♦ Generalized lymphoid hyperplasia	♦ <i>H. influenzae</i> ♦ Streptococci (group A beta hemolytic) ♦ Staphylococci ♦ <i>Moraxella catarrhalis</i> ♦ <i>Mycobacterium tuberculosis</i> (rare)	♦ Recurrent attacks of rhinitis, sinusitis or chronic tonsillitis ♦ Allergy of upper respiratory tract ♦ Low socioeconomic status ♦ Overcrowding, environmental pollution

Clinical Features

- Usually affects children aged between 3-10 years.

	Symptoms	Signs
Nasal	<ul style="list-style-type: none"> ♦ Nasal obstruction <ul style="list-style-type: none"> – Commonest symptom – Leads to mouth breathing, snoring – Interferes with feeding or suckling a child ♦ Persistent nasal discharge (Anterior and postnasal) <ul style="list-style-type: none"> – Wet bubbly nose – Due to choanal obstruction and associated chronic rhinitis ♦ Recurrent sinusitis <ul style="list-style-type: none"> – Chronic maxillary sinusitis due to persistence of nasal discharge and infection ♦ Epistaxis (Due to nose blowing) ♦ Voice change <ul style="list-style-type: none"> – Toneless voice with loss of nasal quality (rhinolalia clausa) 	<ul style="list-style-type: none"> ♦ Mucoïd or mucopurulent nasal discharge ♦ Mucosal congestion and edema ♦ Decreased fogging on both sides on cold spatula test
Aural	<ul style="list-style-type: none"> ♦ Recurrent attacks of earache (Due to infection and blockage of Eustachian tube) ♦ Ear discharge (Due to serous otitis media) ♦ Deafness (Due to blockage of Eustachian tube) 	<ul style="list-style-type: none"> ♦ Retracted or bulging tympanic membrane (depending upon severity) ♦ Conductive hearing loss on tuning fork test ♦ Fluid levels
Throat (Nasopharyngeal)	<ul style="list-style-type: none"> ♦ Recurrent sore throat (Due to mouth breathing) ♦ Painful swallowing (Due to recurrent pharyngitis or tonsillitis) 	<ul style="list-style-type: none"> ♦ Post nasal discharge ♦ Enlarge mass of adenoids on posterosuperior wall of nasopharynx ♦ Mucosal congestion ♦ Granular posterior pharyngeal wall
General	<ul style="list-style-type: none"> ♦ Malnutrition (Due to feeding and breathing cannot occur simultaneously) ♦ Nocturnal enuresis (Due to suffocation because of nasal obstruction) 	<ul style="list-style-type: none"> ♦ Adenoid facies <ul style="list-style-type: none"> – Characteristic facial appearance in long standing case of adenoiditis ♦ Cause <ul style="list-style-type: none"> – Chronic nasal obstruction and mouth breathing ♦ Features <ul style="list-style-type: none"> – Elongated face with dull idiotic expression – Open mouth with mouth breathing – Prominent and crowded upper teeth – Hitched up upper lip – Loss of nasolabial fold – Pinched in appearance of nose (due to disuse atrophy of alae nasi) – Highly arched palate – Deafness ♦ Pulmonary hypertension ♦ Cor pulmonale ♦ Pigeon shaped chest ♦ Growth retardation ♦ Failure to thrive

Investigations

Diagnostic nasal endoscopy	X-rays
♦ To assess degree of hypertrophy and compromise in nasopharyngeal airway	♦ Lateral view of neck reveals size of adenoids and extent of compromised nasopharyngeal air space

Differential Diagnosis

a. Nasal obstruction due to other causes like rhinitis, sinusitis, antrochoanal polyp

- b. Orthodontic abnormality
 - High arched palate with protruding teeth
 - No nasal obstruction
- c. Thornwaldt's disease
 - Seen in adults
 - Midline cyst in nasopharynx because of persistence of median furrow of adenoids.

Treatment

Conservative

Indications	Techniques
<ul style="list-style-type: none"> ♦ Less marked symptoms 	<ul style="list-style-type: none"> ♦ Breathing exercise ♦ Systemic antibiotics to control infection ♦ Decongestant nasal drops and orally to reestablish nasal breathing ♦ Antihistaminics

Operative Techniques

Adenoidectomy

Indications	Contraindications
<ul style="list-style-type: none"> ♦ Adenoid hypertrophy with marked symptoms ♦ Recurrent rhinosinusitis ♦ Aural conditions associated with adenoiditis ♦ Chronic secretory otitis media or benign CSOM causing recurrent ear discharge ♦ Dental malocclusion (prevents recurrence) 	<ul style="list-style-type: none"> ♦ Cleft palate or submucous palate (results in palatopharyngeal insufficiency) ♦ Age under 3 years ♦ Cervical spine pathology ♦ Bleeding diathesis ♦ Acute infection of upper respiratory tract

Anesthesia	Position
<ul style="list-style-type: none"> ♦ General anesthesia with endotracheal intubation 	<ul style="list-style-type: none"> ♦ Supine with extended head in neutral position by placing pillow under shoulders and head stabilized by rubber ring under it (Rose's position)

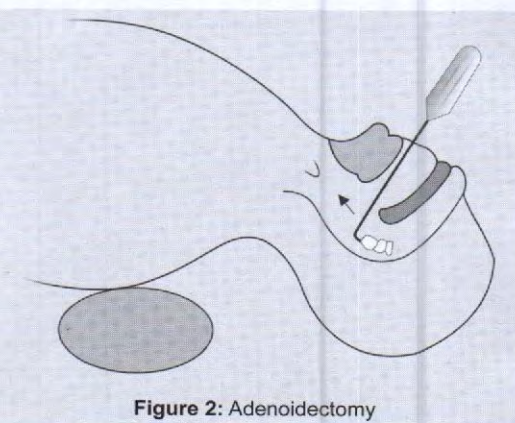


Figure 2: Adenoidectomy

Procedure

- Insert mouth gag
- Examine nasopharynx (by retracting soft palate with curved end of tongue depressor) to confirm diagnosis, assess size of adenoids and push lateral adenoid masses towards midline
- Insert St. Clair Thomson adenoid curette with guard (of proper size) into oropharynx with blade pointing laterally and then rotated 90° to enter nasopharynx
- Bring its free edge in contact with posterior border of nasal septum and then pressed backwards to engage adenoids
- While engaging adenoids slightly flex head to avoid injury to odontoid process
- Shave off adenoids with gentle downward and forward sweeping movement across roof and posterior wall of nasopharynx

- Remove lateral masses with smaller curettes and tags of lymphoid tissue with punch forceps
- Hemostasis achieved by postnasal packing with gauze under pressure or electrocautery in persistent bleeders.

Postoperative care	Complications	
	Immediate	Delayed
<ul style="list-style-type: none"> ♦ Watch for vitals and bleeding from nose and mouth ♦ Antibiotics and analgesics for a week ♦ Maintain oral hygiene ♦ Soft or liquid diet for 2 days and shifting gradually to solid food 	<ul style="list-style-type: none"> i. Hemorrhage <ul style="list-style-type: none"> ♦ Primary hemorrhage <ul style="list-style-type: none"> – Due to partial removal of adenoids – Fills nose and mouth with blood – Usually stops after complete removal or tags and postnasal packing ♦ Reactionary hemorrhage <ul style="list-style-type: none"> – Due to incomplete removal of adenoid tissue or excessive curetting of nasopharynx – Indicated by rising pulse rate – Controlled by postnasal pack, ligation or electrocoagulation of bleeding vessels ii. Injury to surrounding structures <ul style="list-style-type: none"> ♦ Injury to Eustachian tube opening, uvula and soft palate ♦ Injury to pharyngeal musculature (due to undue pressure of curette) ♦ Subluxation of atlantoaxial joint (due to hyperextension of neck) 	<ul style="list-style-type: none"> i. Secondary hemorrhage (rare) <ul style="list-style-type: none"> ♦ Due to infection ♦ Indicated by vomitus of dark colored blood swallowed in postoperative period ♦ Treated by rest and antibiotics ii. Velopharyngeal insufficiency iii. Nasopharyngeal stenosis (due to scarring) iv. Otitis media (due to fibrosis of Eustachian tube opening) v. Chronic nasopharyngitis vi. Nasal voice (rhinolalia aperta) vii. Recurrence (due to growth of remnant adenoid tissue)

8. Meniere's disease.

Refer Question No. 2 December 2008 (RS2).

9. Singer's nodules.

- Singer's nodules are solid non-neoplastic lesions of vocal cords
- Also called laryngeal nodules or vocal nodules or screamer's nodules

Etiopathogenesis

Etiology	Predisposing factors	Pathogenesis
<ul style="list-style-type: none"> ♦ Vocal trauma (speaking in unnatural low tones for prolonged periods or at high intensities) 	<ul style="list-style-type: none"> ♦ Chronic cough ♦ Gastroesophageal disorder 	<p>Vocal cord abuse/misuse</p> <p>⇓</p> <p>Excessive vibration at junction of anterior 1/3rd and posterior 2/3rd of vocal cord</p> <p>⇓</p> <p>Edema and hemorrhage in submucosal space</p> <p>⇓</p> <p>Hyalinization and fibrosis</p> <p>⇓</p> <p>Hyperplasia of overlying epithelium</p> <p>⇓</p> <p>Vocal nodule</p>

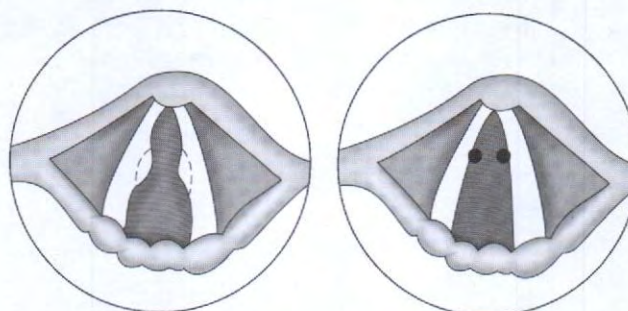
Pathology

Gross appearance	Microscopic examination
<ul style="list-style-type: none"> Soft firm reddish nodules 	<ul style="list-style-type: none"> Nodules covered with normal squamous epithelium Underlying edematous stroma Increased vascularity, dilated blood vessels and hemorrhage Keratoses, acanthosis and parakeratosis of surface epithelium in chronic cases

Clinical Features

- Usually occurs after puberty
- Mostly affects females
- Seen commonly in teachers, actors, hawkers or singers and assertive and talkative individuals

Symptoms	Signs
<ul style="list-style-type: none"> Hoarseness of voice (due to incomplete approximation of cords) Vocal fatigue at end of day Neck pain on prolonged phonation 	<ul style="list-style-type: none"> Symmetrical bilateral swellings at junction of anterior 1/3rd with posterior 2/3rd (area of maximum vibration) Vary from size of pin head to half a pea (rarely >1.5 mm) Soft reddish edematous initially but later becomes grayish or white May be small or may be large and polypoidal May remain firm or may become soft or cystic



Early stage of vocal nodules

Vocal nodules

Figure 3: Singer's nodules**Treatment**

Conservative	Operative
<ul style="list-style-type: none"> Voice rest for several weeks Steroid inhalers (fluticasone propionate) in early cases Speech therapy in early cases Reeducation in voice production to prevent recurrence 	<ul style="list-style-type: none"> Excision of nodule by microlaryngoscopy for large nodules or chronic cases

10. Causes of deafness in child.

Refer Question No. 8 December 2008 (RS2).

11. Vasomotor rhinitis.

Refer Question No. 10 June 2012 (RS2).

12. Tracheostomy tubes.

- Tracheostomy tubes are tube inserted into trachea through tracheostomy incision for providing ventilation

Tracheostomy tubes:

- Tubes inserted into trachea via, tracheostomy incision → ventilation

Parts

a. Outer tube	<ul style="list-style-type: none"> ♦ Fits into tracheostomy opening ♦ May be made up two blades, which when pressed together can be easily introduced into tracheostomy opening without using a pilot and trachea dilator ♦ It may not be split in some tubes
b. Inner tube	<ul style="list-style-type: none"> ♦ Fits snugly into outer tube ♦ Is slightly longer than inner tube so that it can be tracheobronchial secretions may block only projecting end of inner tube without blocking outer tube and inner tube can be cleaned of these secretions and crusts and reinserted without difficulty as often as needed ♦ Has a hole in center so that patient can still have a chance to breath from larynx even when tube is blocked at outer end ♦ Can be fixed to shield of outer tube by lock
c. Shield	<ul style="list-style-type: none"> ♦ Attached to proximal end of outer tube ♦ Used to fix tracheostomy tube to neck by a tape threaded through holes in it
d. Lock	<ul style="list-style-type: none"> ♦ Present in some tubes, meant to fix inner tube into outer tube
e. Pilot or obturator	<ul style="list-style-type: none"> ♦ Blunt ended and curved ♦ Helps in introduction of tube into trachea through tracheostomy incision

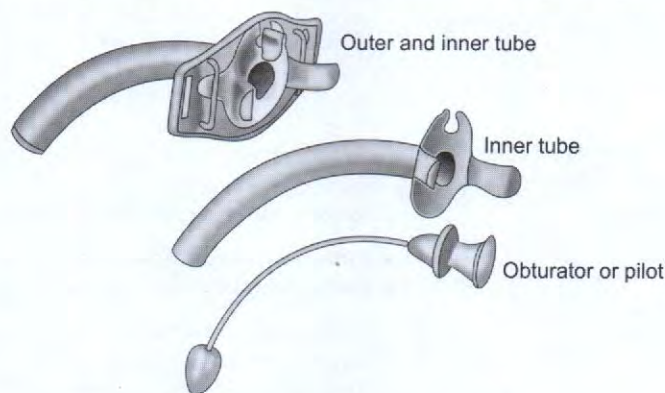


Figure 4: Tracheostomy tube—parts

Types

Based on cuffs	Based on lumen	Based on number of holes	Based on length
(Cuffs prevents aspiration of pharyngeal secretions into trachea and also prevents air leak) <ul style="list-style-type: none"> ♦ <u>Uncuffed and cuffed tube</u> ♦ <u>Double cuff tube</u> <ul style="list-style-type: none"> – Have 2 cuffs that can be alternatively inflated to prevent pressure necrosis at single site 	<ul style="list-style-type: none"> ♦ <u>Single lumen tube</u> <ul style="list-style-type: none"> – No inner cannula ♦ <u>Double lumen tube</u> <ul style="list-style-type: none"> – Have an inner cannula and an outer cannulas – Easier to remove, clean and replace inner cannula 	<ul style="list-style-type: none"> ♦ <u>Unfenestrated tube</u> ♦ <u>Fenestrated tube</u> <ul style="list-style-type: none"> – Have single or multiple holes at upper curvature – Helps in speech or weaning from tracheostomy 	<ul style="list-style-type: none"> ♦ <u>Adjustable flange long tube</u> ♦ <u>Extra long tube</u> indicated in presence of thick and swollen pretracheal tube or to by-pass tracheal growth or stenosis

Materials

Material	Advantages	Disadvantages
a. <u>Silver</u> <ul style="list-style-type: none"> ♦ Fuller, Negus, Jackson's tube 	<ul style="list-style-type: none"> ♦ <u>Non-irritating</u> 	<ul style="list-style-type: none"> ♦ <u>Costly</u>
b. <u>PVC (Portex)</u>	<ul style="list-style-type: none"> ♦ <u>Disposable</u>, single use tubes ♦ <u>Thermolabile</u> and adjusts to tracheal wall 	
c. <u>Silicone</u>	<ul style="list-style-type: none"> ♦ Bacteria and secretions do not adhere ♦ <u>Minimal crusting</u> 	

Contd...

Contd...

Material	Advantages	Disadvantages
d. <u>Siliconised PVC</u>	<ul style="list-style-type: none"> ♦ <u>Thermolabile</u> and adjusts to tracheal wall ♦ <u>Minimal crusting</u> 	
e. <u>Silastic</u>	<ul style="list-style-type: none"> ♦ <u>Soft and non-irritating</u> ♦ <u>Minimal crusting</u> 	
f. <u>Armoured tubes</u> <ul style="list-style-type: none"> ♦ PVC tubes reinforced by spirals or rings of stainless steel 	<ul style="list-style-type: none"> ♦ <u>No easily kinked</u> 	

Size

ISO Sizing system	Ideal size
<ul style="list-style-type: none"> ♦ Tracheostomy are <u>numbered based on inner diameter of outer tube</u> (International Standards Organization (ISO) sizing system) ♦ Size is sometimes expressed in French gauge (Fg) ♦ $Fg = \text{outer diameter} \times \pi$ (3.14) ♦ Size is marked on flanges of tube and also on pilot of balloon 	<ul style="list-style-type: none"> ♦ For adults ♦ Between 6.0-9.0mm (average size male: 9.0 and female 8.0) ♦ For children (3-14 yrs) ♦ Between 5.0-7.0 mm ♦ For children (< 3 yrs) ♦ Between 2.5-4.5 mm

Applications²

Tube	Indication
♦ <u>Cuffed</u> tube with disposable inner cannula	♦ To obtain a closed circuit for <u>ventilation</u>
♦ <u>Cuffless</u> tube with disposable inner cannula	<ul style="list-style-type: none"> ♦ Patients with tracheal problems ♦ Patients who are ready <u>for decannulation</u>
♦ Cuffed tube with <u>reusable inner</u> cannula	<ul style="list-style-type: none"> ♦ To obtain a closed circuit for <u>ventilation</u> ♦ Patients with tracheal problems ♦ Patients who are ready for <u>decannulation</u>
♦ <u>Fenestrated cuffed</u> tracheostomy tube	♦ Patients who are on the ventilator but are not able <u>to tolerate a speaking valve to speak</u>
♦ <u>Fenestrated cuffless</u> tracheostomy tube	♦ Patients who have <u>difficulty using a speaking valve</u>
♦ Metal tracheostomy tube	♦ Not used as frequently anymore

Characteristics of Ideal Tube

- All parts should fit snugly and inner end of inner tube should project slightly beyond outer tube
- Should permit optimum flow of air
- Shorter shaft
- Greater radius of curvature
- Smooth inner surface
- Nontoxic
- Minimum tissue reactivity
- Easily connected to ventilator
- Easy to clean and change.

Ref:

1. Linda L. Morris, M. Sherif Afifi; Tracheostomies : the complete guide (2010); Springer Publishing Company, New York; Pg. 41-113
2. <http://www.hopkinsmedicine.org/tracheostomy/about/types.html> accessed on 29th Feb. 2012.

SHORT ANSWERS**13. Ludwig's angina.**

Refer Question No. 5 December 2011 (RS2).

14. Ototoxicity.

Refer Question No. 5 December 2008 (RS2).

15. Sudden hearing loss.

Refer Question No. 4 June 2017 (RS2).

16. Rhinoscleroma.

Refer Question No. 4 December 2013 (RS2).

17. Nasal polyp.

Refer Question No. 2 December 2013 (RS2).

18. Otitis externa.

Refer Question No. 15 December 2008 (RS2).

19. Cardinal signs of chronic tonsillitis.

Refer Question No. 22 June 2015 (RS2).

20. Draw a diagram right tympanic membrane and label its parts.

Tympanic membrane or eardrum or drumhead is a thin trilaminar partition between external acoustic meatus and middle ear.

Features

- It is oval, translucent and pearly gray measuring 9×10 mm and 0.1 mm thick
- It is placed obliquely at an angle of 55° with floor of meatus thus it faces downward, forward and laterally

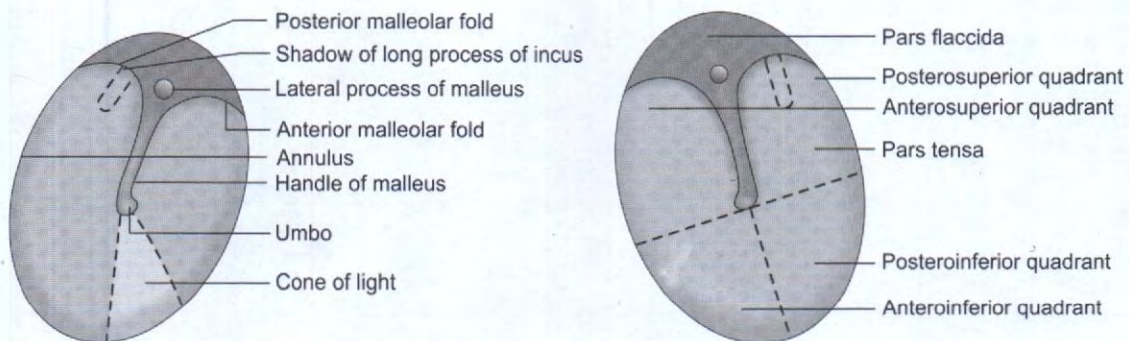


Figure 5: Tympanic membrane
(right side showing parts and left side showing quadrants)

- Membrane has two surfaces – outer and inner
- Outer surface is concave and is lined by skin
- Inner surface is convex and is lined by respiratory mucosa of middle ear
- Inner surface provides attachment to malleus and point of maximum convexity is at tip of malleus and is called umbo
- Membrane is fixed in place by thickened circumference fixed in tympanic sulcus of temporal bone
- This sulcus is deficient superiorly where membrane is attached to tympanic notch
- From ends of notch anterior and posterior malleolar folds are prolonged to lateral process of malleus.

Parts	Layers
<ul style="list-style-type: none"> ♦ Pars tensa is greater part which is tightly stretched ♦ Pars flaccida (Shrapnell's membrane) is part between two malleolar fold and so called because it is loose 	<ul style="list-style-type: none"> ♦ Outer cuticular layer (skin) continuous with skin lining meatus ♦ Middle fibrous layer (absent in pars flaccida) is made up superficial radiating fibers, deep circular fibers and parabolic fibers ♦ Inner mucus layer (low ciliated columnar epithelium) continuous with mucosa of middle ear

	Outer surface	Inner surface
Arterial supply	♦ Deep articular branch of maxillary artery	<ul style="list-style-type: none"> ♦ Tympanic branch of maxillary artery ♦ Posterior branch of stylomastoid branches of the posterior auricular artery
Venous drainage	♦ External jugular vein	♦ Transverse sinus
Nerve supply	<ul style="list-style-type: none"> ♦ Auriculotemporal nerve (Anteroinferior part) ♦ Auricular branch of vagus nerve (Posterosuperior part) 	♦ Tympanic branch of glossopharyngeal nerve

Applied Anatomy

- Any incision of tympanic membrane (myringotomy) should be made in posteroinferior quadrant to avoid injury to chorda tympani nerve and ossicles of ear.

21. Schwabach's test.

Refer Question No. 18 December 2007 (RS2).

22 . Laryngeal web.

- Laryngeal web is formation of fibrous tissue covered by epithelium between anterior parts of vocal cords.

Etiopathogenesis	Site of web
<ul style="list-style-type: none"> ♦ Arrest of laryngeal development at 10th week of fetal life resulting in incomplete recanalization of larynx 	<ul style="list-style-type: none"> ♦ Glottis level (75%) ♦ Supraglottic level (25%)

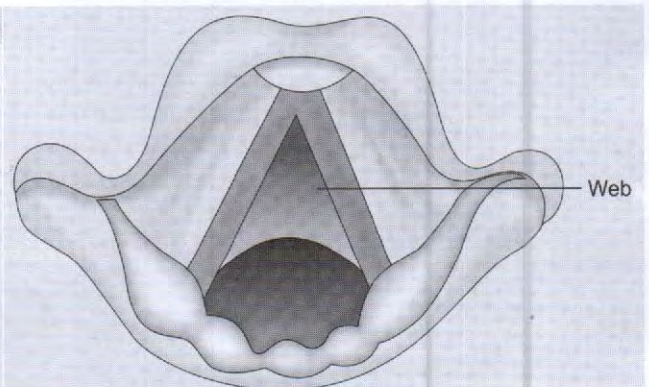


Figure 6: Laryngeal web

<i>Clinical features (Manifests from birth)</i>		<i>Treatment</i>	
<i>Symptoms</i>	<i>Signs</i>	<i>Conservative</i>	<i>Operative</i>
<ul style="list-style-type: none"> ♦ Asymptomatic (if small web) ♦ Stridor (inspiratory) ♦ Weak cry ♦ Aphonia 	<ul style="list-style-type: none"> ♦ White or pink, thick or thin membrane attached anteriorly to both vocal cords with sharp and concave posterior margin 	<ul style="list-style-type: none"> ♦ Reassurance for milder form 	<ul style="list-style-type: none"> ♦ Tracheostomy for severe stridor and dyspnea ♦ Serial microlaryngoscopic dilatation for thin webs ♦ Excision via laryngofissure and placement of McKnight's silicone keel to prevent synechia until cordal reepithelialization occurs for thick webs

MBBS PHASE III EXAMINATION

DECEMBER 2016

(Revised Scheme 2 & 3)

■ LONG ESSAYS

1. Discuss the etiology, clinical features and management of acute coalescent mastoiditis.

Refer Question No. 3 December 2009 (RS2).

2. Discuss the etiology, clinical features and management of juvenile nasopharyngeal angiofibroma.

Refer Question No. 1 December 2009 (RS2).

■ SHORT ESSAYS

3. Le Fort fractures.

Refer Question No. 3 December 2010 (RS2).

4. Management of Meniere's disease.

Refer Question No. 2 December 2008 (RS2).

5. Otogenic causes of facial palsy.

Refer Question No. 11 December 2008 (RS2).

6. Indications and complications of tracheostomy.

Refer Question No. 9 December 2007 (RS2).

7. Clinical features and management of peritonsillar abscess.

Refer Question No. 7 June 2011 (RS2).

8. Differential diagnosis of lateral neck swellings.

Refer Question No. 8 June 2011 (RS2).

9. Foreign body in bronchus.

Refer Question No. 9 June 2011 (RS2).

10. Theories of hearing.

Refer Question No. 1 December 2007 (RS2).

11. Anatomy of the middle ear cleft.

Refer Question No. 1 June 2012 (RS2).

12. Clinical features and management of septal perforation.

Refer Question No. 12 June 2011 (RS2).

■ SHORT ANSWERS**13. Rhinophyma.**

Refer Question No. 13 June 2011 (RS2).

14. Vocal polyp.

Refer Question No. 22 June 2008 (RS2).

15. Ranula.

Refer Question No. 15 June 2011 (RS2).

16. Branchial cyst.

Refer Question No. 16 June 2011 (RS2).

17. Exostosis in external auditory canal.

Refer Question No. 17 June 2011 (RS2).

18. Rinne's test.

Refer Question No. 18 December 2007 (RS2).

19. Rhinitis sicca.

Refer Question No. 19 June 2011 (RS2).

20. Little's area.

Refer Question No. 20 June 2011 (RS2).

21. Waldeyer's ring.

Refer Question No. 3 June 2015 (RS2).

22. Reinke's edema.

Refer Question No. 20 June 2010 (RS2).

MBBS PHASE III EXAMINATION

JUNE 2017

(Revised Scheme 2 & 3)

LONG ESSAYS

1. Describe the etiology, pathology, clinical features and management of cholesteatoma.

Refer Question No. 1 June 2013 (RS2).

2. Describe the etiology, clinical features and management of carcinoma maxilla.

Maxillary carcinoma is most frequent of uncommon paranasal air sinuses malignancies

<i>Etiology</i>	<i>Predisposing factors</i>	<i>Pathology</i>
<ul style="list-style-type: none">♦ Chronic sinusitis♦ Protracted polyposis♦ Smoking♦ Snuff (in Bantu tribe of South Africa)	<ul style="list-style-type: none">♦ Atmospheric pollution♦ Wood dust (ebony wood)♦ Nickel dust	<p>Most common</p> <ul style="list-style-type: none">♦ Squamous cell carcinoma (80%) <p>Rare</p> <ul style="list-style-type: none">♦ Adenocarcinoma♦ Basal cell carcinoma♦ Melanoma♦ Sarcoma

Classification

Ohngren's classification

- ♦ Divides tumor into infrastructure and suprastructure groups by drawing an imaginary line joining inner canthus of eye to angle of mandible



Figure 1: Ohngren's line

Lederman's classification

- ♦ Divides tumor into suprastructure, mesostructure and infrastructure groups by 2 horizontal imaginary lines of Seibilleau passing through floor of orbit and floor of maxillary antrum

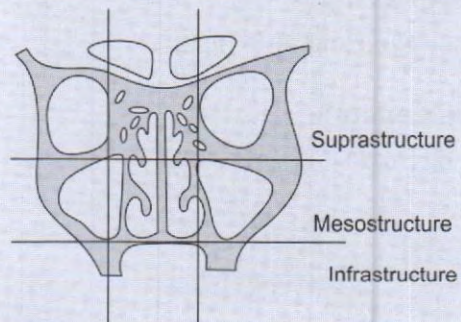


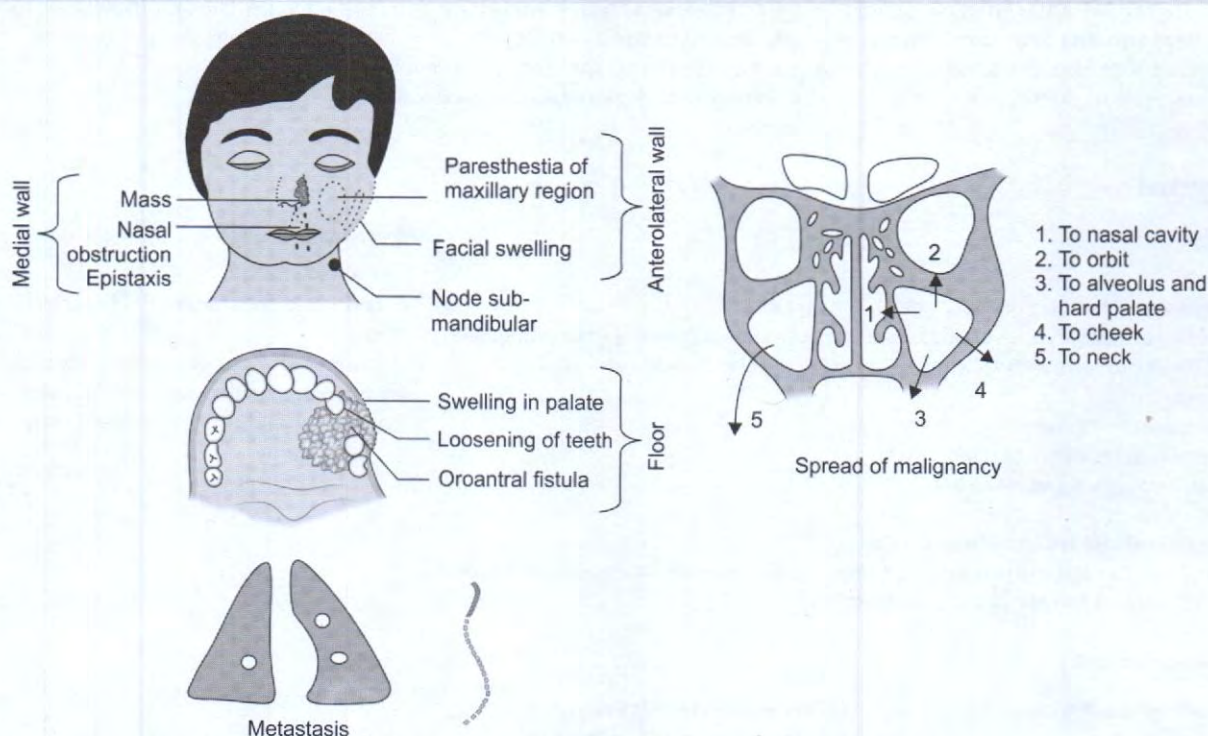
Figure 2: Lederman's classification

Clinical Features

- Usually occurs between age of 40 to 70 years
- Frequently affects males

A. Early stage

Symptoms (similar to sinusitis)	Signs
<ul style="list-style-type: none"> ♦ Dull pain over maxillary sinus which increases on bending forward and coughing (earliest symptom) ♦ Watering on eye of affected side ♦ Blood stained, painless nasal discharge ♦ Intermittent and progressive nasal obstruction on affected side ♦ Toothache ♦ Anesthesia over malar region 	<ul style="list-style-type: none"> ♦ Infraorbital anesthesia (early sign) ♦ Obliteration of canine fossa with softening in area

**Figure 3:** Early stage of carcinoma maxilla**B. Late stage (features of spread of tumor)**

	Symptoms	Signs
Anterior spread	♦ Swelling of maxillary region due to subcutaneous infiltration	♦ Swelling on cheek with fungation
Superior spread	<ul style="list-style-type: none"> ♦ Ocular pain ♦ Diplopia with proptosis of eyeball ♦ Loss of vision (later) 	<ul style="list-style-type: none"> ♦ Obliteration of infraorbital margin ♦ Proptosis with diplopia ♦ Fixation of eye ♦ Loss of vision
Medial spread	<ul style="list-style-type: none"> ♦ Blocking of nose on affected side due to protrusion of granulomatous mass ♦ Blood stained nasal discharge ♦ Parosmia and anosmia 	<ul style="list-style-type: none"> ♦ Bulging of lateral wall of nose ♦ Filling of nasal cavity with granulomatous mass which bleeds on touch

Contd....

	Symptoms	Signs
Inferior spread	<ul style="list-style-type: none"> ♦ Painless fall of teeth ♦ Swelling in oral cavity ♦ Denture become illfitting 	<ul style="list-style-type: none"> ♦ Oroantral fistula in gingivolabial groove ♦ Bulging of hard palate ♦ Absence of teeth ♦ Cauliflower-like mass in oral cavity
Posterior spread	<ul style="list-style-type: none"> ♦ Trismus (due to involvement of pterygoid muscle) ♦ Infraorbital neuralgia 	<ul style="list-style-type: none"> ♦ Trismus
Others	<ul style="list-style-type: none"> ♦ Cachexia (in advanced cases) 	<ul style="list-style-type: none"> ♦ Enlargement of submandibular lymph nodes due to metastases

Spread

Local spread	Lymphatic spread	Blood borne spread
<ul style="list-style-type: none"> ♦ May spread to adjacent anatomical structures involving orbit, cribriform plate, alveolus, palate, skin, infratemporal region and pterygoid muscles 	<ul style="list-style-type: none"> ♦ Retropharyngeal lymph nodes are usually affected first but difficult to detect ♦ Submandibular lymph nodes are involved following involvement of skin, anterior wall and alveolus 	<ul style="list-style-type: none"> ♦ Occurs late and metastases to lungs (most common) and bone

Investigations

Radiology	Histopathology
i. X-ray <ul style="list-style-type: none"> ♦ Generalized haziness or opacity patch in early cases ♦ Erosion of maxillary sinus boundaries and opacification of maxillary sinus with spread of opacity to surrounding tissues in late stage (diagnostic feature) ii. CT scan <ul style="list-style-type: none"> ♦ Investigation of choice ♦ Demonstrates extent of tumor spread ♦ Also helps in staging of disease iii. MRI <ul style="list-style-type: none"> ♦ Helps distinguish tumors from sinusitis ♦ Shows better soft tissue delineation thus helps in assessing involvement of orbit, infratemporal and pterygopalatine fossa 	i. Exfoliative cytology <ul style="list-style-type: none"> ♦ Performed on returning fluid of antral lavage ii. Biopsy <ul style="list-style-type: none"> ♦ Performed through antroscopy (sinuscopy) or by opening maxillary antrum by Caldwell Luc operation in early cases and directly in late cases

Differential Diagnosis

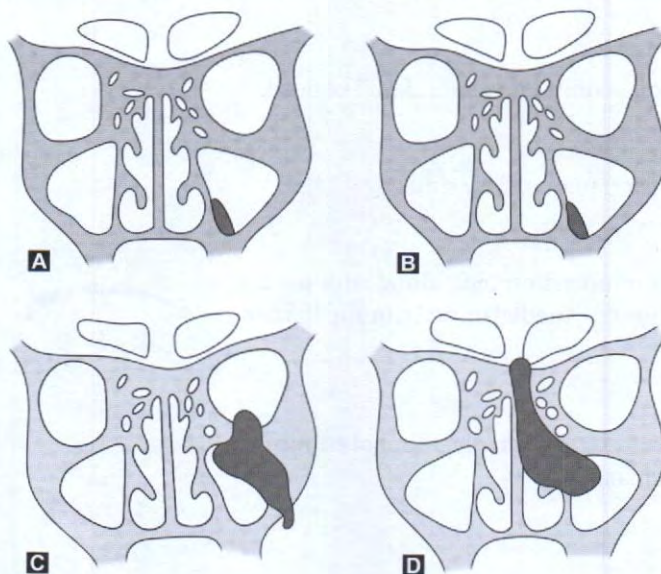
a. Hypertrophic turbinates	<ul style="list-style-type: none"> ♦ Pink, tender and soft to touch ♦ Bony turbinates felt on deeper touch ♦ Shrink with decongestant
b. Rhinosporidiosis	<ul style="list-style-type: none"> ♦ Mulberry like mass ♦ Bleeds readily on touching ♦ Seen in patients from coastal areas
c. Angioma of nasal septum	<ul style="list-style-type: none"> ♦ Single, red, smooth mass which bleed readily on touch
d. Alveolar abscess	<ul style="list-style-type: none"> ♦ Painful tooth ♦ Swelling of alveolar margin and face
e. Cellulitis of cheek	<ul style="list-style-type: none"> ♦ Secondary to cellulitis of nose

TNM Staging

Stage	Tumor (T)	Nodal involvement (N)	Metastasis (M)
X	—	Cannot be assessed	Cannot be assessed
0	—	No metastasis	No distant metastasis

Contd....

Stage	Tumor (T)	Nodal involvement (N)	Metastasis (M)
1	Limited to maxillary sinus with no erosion or destruction of bone	Single ipsilateral node, 3 cm or less	Distant metastasis
2a	Bone erosion or destruction including extension into hard palate and/or middle nasal meatus, except extension to posterior wall of maxillary sinus and pterygoid plates	Single ipsilateral node, 3-6 cm	—
2b		Multiple ipsilateral lymph nodes, <6 cm	—
2c		Bilateral or contralateral node, <6 cm	—
3	Invasion of bone of posterior wall of maxillary sinus, subcutaneous tissues, floor or medial wall of orbit, pterygoid fossa and ethmoid sinuses	Nodes >6 cm	—
4a	Invasion of anterior orbital contents, skin of cheek, pterygoid plates, infratemporal fossa, cribriform plate, sphenoid or frontal sinuses	—	—
4b	Invasion of orbital apex, dura mater, brain, middle cranial fossa, cranial nerves other than maxillary division of trigeminal nerve, nasopharynx or clivus	—	—



Figures 4A to D: TNM classification (A) T1—tumor confined to infrastructure without bony erosion; (B) T2—tumor confined to infrastructure with bony erosion; (C) T3—tumor invading orbit or cheek; (D) T4—tumour involving cribriform plate, posterior ethmoids, nasopharynx

Grading

Grade	Tumor (T)	Nodal involvement (N)	Metastasis (M)
I	1	0	0
II	2	0	0
III	3	0	0
	1/2/3	1	0
IVA	4a	0	0
	4b	1	0
IVB	1/2/3/4	2/3	0
IVC	1/2/3/4	0/1/2/3	1

Treatment**Conservative**

Radiotherapy	Chemotherapy
Indications <ul style="list-style-type: none"> ♦ Early cases ♦ Preoperative to reduce size and vascularity ♦ Post-operative Dose <ul style="list-style-type: none"> ♦ 5000-6000 rads of cobalt-60 over a period of 5-6 weeks Complications <ul style="list-style-type: none"> ♦ Damage to eyes and parotid gland ♦ Necrosis of skin flaps (if surgery is performed) 	Indications <ul style="list-style-type: none"> ♦ Early cases ♦ Preoperative to reduce size and vascularity Drugs <ul style="list-style-type: none"> ♦ Methotrexate ♦ 5-fluorouracil ♦ Cisplatin Route of administration <ul style="list-style-type: none"> ♦ Local perfusion through maxillary artery

Operative

- Depends upon type of cancer and extent of involvement

Techniques

a. Median maxillectomy

Indications

- Early malignancy of maxilla confined to lateral wall of nose
- Inverted papilloma
- Columnar cell papilloma
- Angiofibroma with extension to maxillary sinus
- Extensive fungal sinusitis.

Principle

- Medial part of maxilla is removed en bloc along with lateral wall of nose including ethmoids, medial orbital rim and lamina papyracea.

b. Total maxillectomy

Indications

- T2 and T3 tumors of maxilla without involvement of ethmoids
- Radioresidual tumors of maxilla

Contraindications

- Distant metastases
- Poor general conditions
- Nasopharyngeal involvement
- Intracranial involvement

Anesthesia

- General

Incision

- Weber-Ferguson incision

Procedure

- Following incision, expose facial surface of maxilla by elevating skin and soft tissues
- Separate soft palate and divide hard palate in center
- Break bony attachments of maxilla and remove maxilla.

c. Extended maxillectomy

Indications

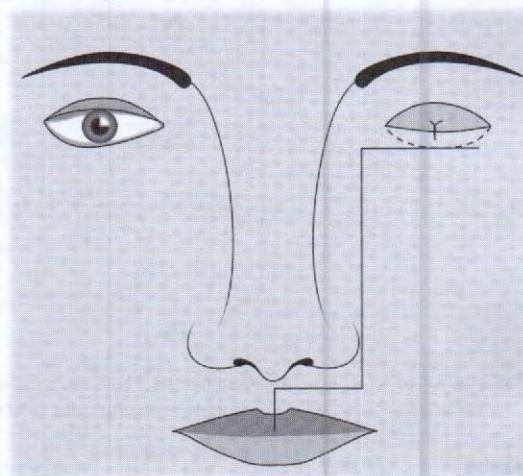
- Extension of tumor to orbit, skin and infratemporal fossa

Principle

- Maxillectomy along with orbital exenteration, excision of skin, face or soft tissue of infratemporal fossa

Prognosis

- 5 years survival rate in 30-40% cases when tumor is confined within maxillary bone.

**Figure 5:** Total maxillectomy

■ SHORT ESSAYS

3. Etiology and management of malignant otitis externa.

Refer Question No. 11 December 2010 (RS2).

4. Causes and management of sudden sensorineural hearing loss.

- Sudden hearing loss is sensorineural hearing loss developed over a period of hours or within 3 days.

Etiology (in the Very Inner Ear Too, No Major Pathology)

a. Idiopathic (most common)	
b. Trauma	<ul style="list-style-type: none"> ♦ Head injury with fracture of petrous part of temporal bone ♦ Iatrogenic (Ear operations) ♦ Barotrauma ♦ Noise induced ♦ Spontaneous rupture of cochlear membrane
c. Vascular	<ul style="list-style-type: none"> ♦ Hemorrhage in labyrinth (trauma, hypertension, leukemia, purpura, etc.) ♦ Vascular pathology of labyrinthine or cochlear artery (embolism, thrombosis or vasospasm)
d. Infections	<ul style="list-style-type: none"> ♦ Mumps ♦ Herpes zoster ♦ Meningitis ♦ Encephalitis ♦ Syphilis ♦ Otitis media
e. Ear	<ul style="list-style-type: none"> ♦ Meniere's disease ♦ Cogan's syndrome ♦ Large vestibular aqueduct
f. Toxic	<ul style="list-style-type: none"> ♦ Ototoxic drugs ♦ Insecticides
g. Neoplastic	<ul style="list-style-type: none"> ♦ Acoustic neuroma ♦ Carcinomatous neuropathy ♦ Metastasis in cerebellopontine (CP) angle
h. Miscellaneous	<ul style="list-style-type: none"> ♦ Multiple sclerosis ♦ Hypothyroidism ♦ Sarcoidosis ♦ Raised intracranial pressure
i. Psychogenic	

Clinical Features

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Sudden hearing loss within period of hours or few days ♦ Loss may be partial or complete ♦ Mostly unilateral ♦ Accompanied by tinnitus or temporary spell of vertigo 	<ul style="list-style-type: none"> ♦ Deterioration of >35 dB in at least 3 adjacent frequencies within 3 days

Investigations

a. Audiometry	
b. Vestibular tests	
c. Pathological tests	For syphilis, diabetes, hypothyroidism, blood disorders and lipid profile
d. Radiological studies	X-ray, CT and MRI of temporal bone

Treatment**Supportive**

a. Bed rest	♦ In head raised position
b. Sedatives	♦ To relieve anxiety and associated giddiness
c. Steroid therapy	♦ Prednisolone 15 mg TID dose for 10 days followed by tailing off ♦ Helps by relieving edema
d. Inhalation of carbogen (95% O ₂ + 5% CO ₂)	♦ Improves oxygenation by increasing cochlear blood flow
e. Hyperbaric oxygen therapy	♦ Beneficial if given within 1st month of onset
f. Low molecular weight dextran	♦ Decreases viscosity of blood
g. Vasodilator drugs	♦ Histamin IV in first 3 days ♦ Nylidrin hydrochloride, nicotinic acid orally or parenterally
h. Stellate ganglion block	♦ To relieve vasospasm by correcting autonomic balance

Specific

- Treatment of underlying cause.

Prognosis

- Spontaneous recovery in 50% patients within 15 days
- Poor prognosis after 1 month.

5. Etiology and management of nasal polyposis.

Refer Question No. 2 December 2013 (RS2).

6. Describe in detail the surgical approaches to nasopharynx.

<i>Surgical difficult to assess nasopharynx</i>	<i>Ideal surgical approach</i>	<i>Criteria to choose surgical approach</i>
♦ Central location ♦ Surrounding facial skeleton, skull base ♦ Presence of great vessels and lower cranial nerves	♦ Provide adequate exposure to nasopharynx for tumor resection ♦ Great vessels must be safely controlled ♦ Lower cranial nerves should be spared - a difficult task indeed	♦ Extent of tumor ♦ Surgical expertise ♦ Facilities available

Surgical Approaches

Anterior approaches	
a. Lateral rhinotomy	♦ Well exposes nasal cavity and choana ♦ Can be used alone or in combination with other approaches to enhance nasopharynx exposure Application ♦ Useful in resection of anteriorly placed tumors
b. Transnasal transmaxillary approach	♦ Combination of lateral rhinotomy with medial/subtotal maxillectomy ♦ Exposes nasopharynx, ipsilateral sphenothmoidal complex, pterygopalatine fossa and medial end of infratemporal fossa
c. Midfacial degloving approach	♦ Bilateral transnasal, transmaxillary approach performed via sublabial incision ♦ Exposes pterygopalatine fossa and medial end of infratemporal fossa Advantages ♦ Avoids facial scar ♦ Safeguards infraorbital nerves on both sides

d. Lefort I osteotomy	<ul style="list-style-type: none"> ◆ Transverse maxillary osteotomy through both maxillary sinuses through a sublabial incision allowing whole hard palate and both inferior maxillae to be down fractured ◆ Exposes central skull base and nasopharynx <p>Advantages</p> <ul style="list-style-type: none"> ◆ No facial scars
e. Maxillary swing approach	<ul style="list-style-type: none"> ◆ Most common approach ◆ Exposes nasopharynx and surrounding areas from anterolateral aspect ◆ Through Weber Ferguson incision, maxilla is separated from its bony attachments and swung laterally intact with masseter muscle and cheek flap ◆ Access to opposite side can be established by removing the posterior portion of nasal septum ◆ After tumor resection, maxilla is swung back and fixed to facial skeleton
Inferior approaches	
a. Transpalatal approach	<ul style="list-style-type: none"> ◆ Nasopharynx is accessed by raising palatal mucoperiosteal flap off the hard palate, separating soft palate from its bony portion and removing posterior edge of bony hard palate as needed ◆ Greater palatine neurovascular bundle are mobilized bilaterally to prevent flap necrosis
b. Mandibular swing approach	<ul style="list-style-type: none"> ◆ Combination of transcervical, transmandibular, transpalatal approach via Frazier incision ◆ Soft tissues including parotid gland are elevated from mandible ◆ Midportion of ascending ramus of mandible including the coronoid process is cut and removed to facilitate exposure and to prevent postoperative trismus ◆ Lateral and medial pterygoid muscles are divided to enter the nasopharynx ◆ Tracheostomy is a must to secure the airway ◆ Dead space after tumor removal needs to be repaired
Lateral approach	
a. Infratemporal fossa approach	<ul style="list-style-type: none"> ◆ Approach is via infratemporal fossa <p>Disadvantages</p> <ul style="list-style-type: none"> ◆ Limited by facial nerve and carotid sheath <p>Application</p> <ul style="list-style-type: none"> ◆ Used when tumor extends laterally to involve parapharyngeal space

7. Describe the anatomy of nasal septum. Discuss septal surgeries.

Nasal Septum

- Nasal septum is median osteocartilaginous partition between two halves of nasal cavity
- On each side, it is covered by mucous membrane and forms medial wall of both nasal cavities

Constituents (bones taking part)—*priyanka verma could never rise at morning parade*

- ◆ Perpendicular plate of ethmoid
- ◆ Vomer
- ◆ Crest of nasal bone
- ◆ Nasal spine of frontal bone
- ◆ Rostrum of sphenoid
- ◆ Anterior nasal spine of maxilla
- ◆ Maxillary crest
- ◆ Palatine crest

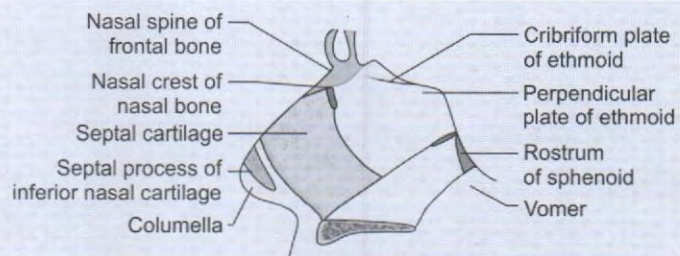


Figure 6: Nasal septum—constituents

Parts

- Bony part
- Cartilaginous part
- Cuticular part which is the lower and the anterior end lined by the skin.

- Border – Superior, anterior, inferior and posterior
- Surfaces – Right and left

Arterial supply

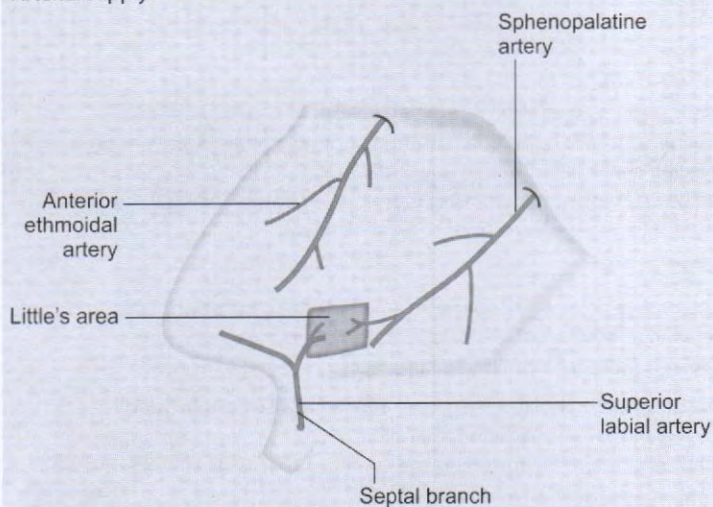


Figure 7: Arterial supply of nasal septum

a. Anterosuperior part by:

- ♦ Anterior ethmoidal artery
- ♦ Superior labial branch of facial artery

b. Posteroinferior part by:

- ♦ Sphenopalatine artery
- ♦ Terminal part of greater palatine artery

Venous drainage

- ♦ Into a plexus in lower part of septum
- ♦ From there, anteriorly into facial vein and posteriorly into pterygoid venous plexus through sphenopalatine vein

Nerve supply

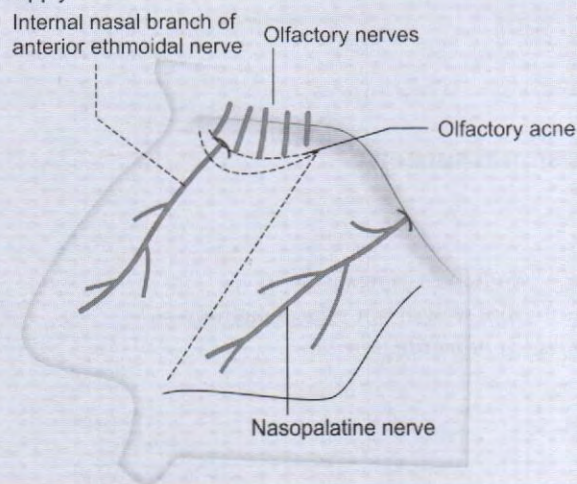


Figure 8: Nerve supply of nasal septum

a. General sensory (Branches of trigeminal nerve)

i. Anterosuperior part

- ♦ Internal nasal branch of anterior ethmoidal nerve

ii. Posteroinferior part

- ♦ Nasopalatine branch of pterygopalatine ganglion

b. Special sensory

- ♦ Olfactory nerve confined to upper part

Lymphatic drainage

a. Anterior half

- ♦ Submandibular nodes

b. Posterior half

- ♦ Retropharyngeal and deep cervical nodes

Applied Anatomy

- Little's area on septum is a commonest site for epistaxis
- Pathological deviation of the nasal septum is often responsible for repeated attacks of common cold, allergic rhinitis, sinusitis.

For Septal Surgeries

Refer Question No. 12 June 2014 (RS2).

8. Discuss the types, procedure and complications of tracheostomy.

Refer Question No. 9 December 2007 (RS2).

9. Discuss the management of an adult patient presenting with hoarseness of voice.

Refer Question No. 5 December 2007 (RS2).

10. Discuss the differential diagnosis of a membranous lesion over the tonsil.

Refer Question No. 12 December 2011 (RS2).

11. Describe the anatomy of parapharyngeal space and its surgical approaches.

Refer Question No. 2 December 2009 (RS2).

12. Indications, contraindications and complications of Bronchoscopy.

Refer Question No. 6 December 2011 (RS2).

■ SHORT ANSWERS**13. Caloric tests.**

Refer Question No. 3 June 2009 (RS2).

14. Acoustic reflex tests.

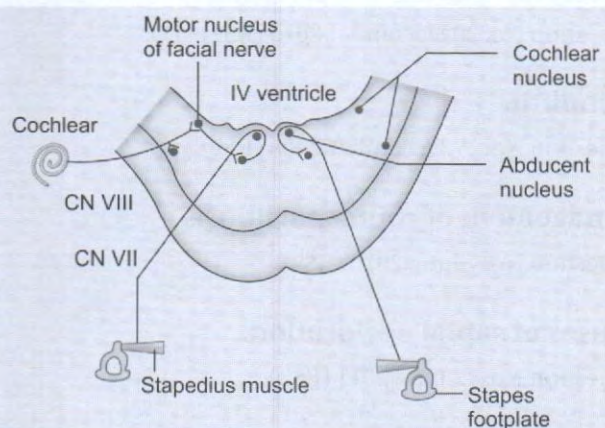
- Acoustic reflex test is an objective audiometry

Principle

- Loud sound (70-100 dB above threshold of hearing) causes bilateral contraction of stapedial muscles which can be detected by tympanometry

Reflex arc

<i>Ipsilateral</i>	<i>Contralateral</i>
CN VIII ↓ Ventral cochlear nucleus ↓ CN VII nucleus ↓ Ipsilateral stapedius muscle	CN VIII ↓ Ventral cochlear nucleus ↓ Contralateral medial superior olivary nucleus ↓ Contralateral CN VII nucleus ↓ Contralateral stapedius muscle

**Figure 9: Reflex arc****Procedure**

Loud tone is delivered to one ear and reflex is picked from same or contralateral ear.

Observation

<i>Findings</i>	<i>Interpretation</i>
♦ Stapedial reflex positive at 70 dB	♦ Normal
♦ Stapedial reflex positive at lower intensities (40-60 dB)	♦ Cochlear type of hearing loss (recruitment)
♦ Stapedial reflex decay (Reflex amplitude reduced to 50% on sustained delivery of 500-1000 Hz tone at 10 dB above acoustic reflex threshold)	♦ VIII nerve lesion (abnormal adaptation)
♦ Absence of stapedial reflex with normal hearing	♦ VIII nerve lesion proximal to nerve to stapedius
♦ Presence of ipsilateral reflex but absence of contralateral reflex	♦ Lesion of crossed pathway in brainstem

Applications

- a. To test hearing in infants and young children (an objective method)
- b. Detect malingers
 - Malingers shows positive stapedial reflex even if there is no response on pure tone audiometry
- c. Detect cochlear pathology
 - Stapedial reflex at lower intensities (40–60 dB) indicates recruitment and cochlear type of hearing loss
- d. Detect VIII nerve lesion
 - Stapedial reflex decay (50% reflex amplitude in response to sustained tone of 500-1000 Hz, delivered 10 dB above acoustic reflex threshold, for a period of 10 sec)
- e. Detect lesions of facial nerve and its prognosis
 - Absence of stapedial reflex in presence of normal hearing indicates lesion of the facial nerve, proximal to nerve to stapedius
 - Appearance of reflex, after it was absent, indicates return of function and favorable prognosis
- f. Detect lesion of brainstem
 - Presence of ipsilateral reflex but absence of contralateral reflex suggests lesion is in the area of crossed pathways in brainstem.

15. Management of otomycosis.

Refer Question No. 10 June 2009 (RS2).

16. Management of BPPV (Benign Paroxysmal Positional Vertigo).

Refer Question No. 20 December 2015 (RS2).

17. Rhinolith.

Refer Question No. 17 December 2008 (RS2).

18. Management of rhinosporidiosis.

Refer Question No. 7 June 2010 (RS2).

19. Causes of septal perforation.

Refer Question No. 12 June 2011 (RS2).

20. Indications for Caldwell Luc operation.

- Caldwell Luc operation is surgical process of opening maxillary antrum through canine fossa by sublabial approach (to avoid external scar) followed by antrostomy for permanent drainage
- Also called radical antrostomy or canine fossa antrostomy.

Indications			Contraindications
Nasal and paranasal	Dental	As an approach	
<ul style="list-style-type: none"> Chronic maxillary sinusitis with irreversible damage to sinus mucosa (not responding to at least 3 antral punctures) Removal of foreign body (like bullet) or part of a tooth displaced during dental procedure Suspected maxillary tumor (for biopsy or resection or insertion of radioactive needles) Recurrent antrochoanal polyp Atrophic rhinitis (for implantation of Stenson's duct into maxillary antrum – Wittmaak's operation or implantation of maxillary sinus mucosa into nasal cavity) Fracture of maxilla or blow out fracture of orbit (for reduction and packing sinus) Hemoantrum 	<ul style="list-style-type: none"> Dental and dentigerous cysts Oroantral fistula 	<ul style="list-style-type: none"> For Horgan's transantral ethmoidectomy To pterygopalatine fossa for maxillary artery ligation, vidian neurectomy, maxillary neurectomy To sphenoid sinus and pituitary gland for hypophysectomy For orbital decompression in malignant exophthalmos For elevation of fractured orbital floor 	<ul style="list-style-type: none"> Age below 17 years (interferes with dentition and growth of face) Acute infections Bleeding disorders General diseases like diabetes, hypertension

Anesthesia

- General with cuffed endotracheal tube and pharyngeal pack to prevent aspiration of blood and secretions.

Position

- Supine with face slightly turned to opposite side on a reclining table with raised head end.

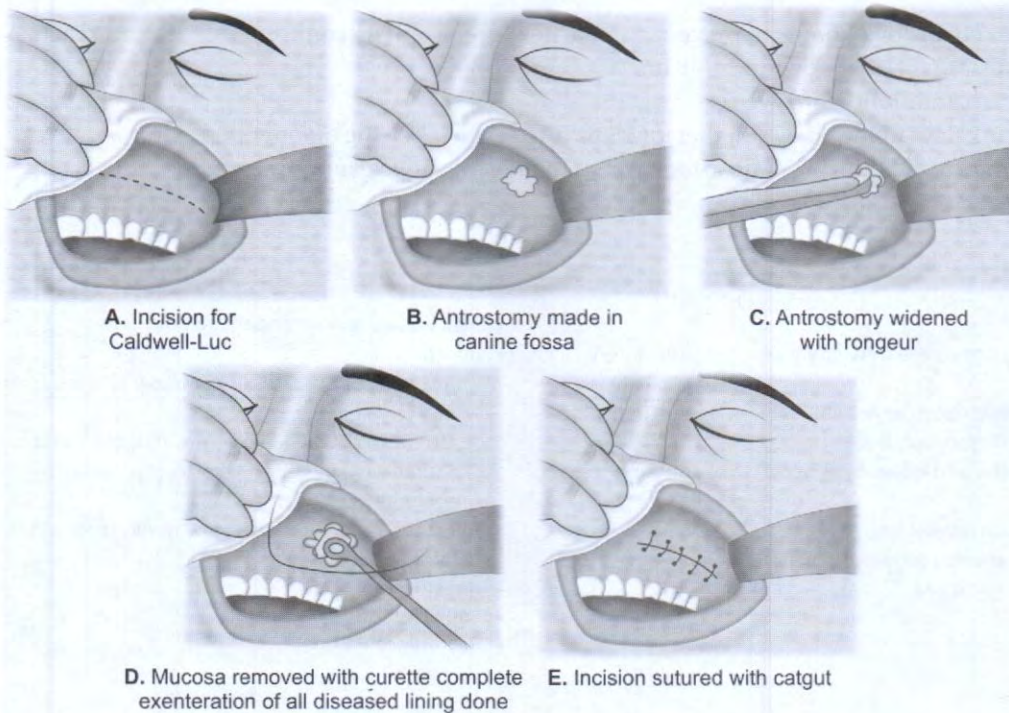


Figure 10: Caldwell Luc's operation

Procedure

- a. Hemostasis
 - Infiltrate labial tissue with 2% lignocaine with 1:100,000 adrenaline
- b. Incision
 - Bone deep horizontal incision cutting through mucosa and periosteum from lateral border of lateral incisor to 2nd molar below gingivolabial sulcus with its end directed upwards
- c. Elevation of flap
 - Raise mucoperiosteal flap using periosteum elevator from canine fossa superolaterally till infraorbital foramen avoiding injury to infraorbital nerve
- d. Opening of antrum
 - Make a hole in anterior wall of antrum using cutting burr or gouge and hammer followed by enlarging it to 1.5-2 cm using Kerrison's sphenoid punch forceps
- e. Dealing with pathology
 - Antral mucosa is inspected and underlying pathology is dealt accordingly
 - Diseased mucosa removed using elevators, curettes and Luc forceps
 - Cysts, benign tumors, foreign body or polyps removed
- f. Antrostomy
 - Make a nasoantral window at lowest and most anterior part of medial wall of sinus by pushing a curved haemostat through inferior meatus into antrum and enlarging this hole to 1.5 cm using Kerrison's and side biting forceps
- g. Packing antrum
 - Pack maxillary antrum with ribbon gauze impregnated with liquid paraffin or furacin and bring out its ends from nasoantral window into nose
 - Also pack nasal cavity on operated side
- h. Closure of wound
 - Suture sublabial incision using 1-2 sutures of 4-0 vicryl.

Postoperative Care

- Ice packs over cheek for 24 hours to prevent edema, hematoma and discomfort
- Remove antral and nasal packing in 24-48 hrs
- Broad spectrum antibiotics for 1 week
- Liquid diet for 2 days followed by soft diet for 15 days (to avoid chewing on operated side)
- Advise patient to avoid blowing of nose for 2 weeks to prevent surgical emphysema.

Complications

Immediate	Delayed
i. Primary hemorrhage <ul style="list-style-type: none"> ♦ Controlled by pressure, adrenaline swab or electrocautery 	♦ Reactionary and secondary hemorrhage
ii. Injuries <ul style="list-style-type: none"> ♦ Damage to infraorbital nerve if soft tissues elevated from anterior wall of antrum upto high level ♦ Damage to orbit and infraorbital nerve while curreting thin roof of antrum ♦ Injury to nasolacrimal duct ♦ Injury to lips, cheek, tongue and teeth roots 	♦ Infection <ul style="list-style-type: none"> – Infection of nose, other paranasal sinuses, middle ear, pharynx and larynx – Orbital infection and osteomyelitis of maxilla (rare) – Pulmonary infection due to aspiration of tracheobronchial secretions
iii. Anesthetic complications	♦ Anesthesia of cheek lasting few weeks to month (due to stretching of infraorbital nerve)
	♦ Anesthesia of teeth
	♦ Sublabial fistula
	♦ Recurrence

21. Treatment of salivary calculi.

- Salivary calculi (also termed as sialolithiasis) is formation of stone in salivary gland or their ducts

<i>Etiology</i>	<i>Common sites</i>	<i>Composition</i>
<ul style="list-style-type: none"> Deposition of calcium phosphate on organic matrix of mucin or cellular debris 	<ul style="list-style-type: none"> Submandibular gland or its duct (Wharton's duct) (because of tortuous course and secretion rich in mucoid and calcium) (90%) Parotid gland or its duct (10%) 	<ul style="list-style-type: none"> Organic matrix with inorganic crystalline body carbohydrate and phosphates of calcium and magnesium

<i>Clinical features</i>		
<i>Symptoms</i>	<i>Signs</i>	<i>Investigations</i>
<ul style="list-style-type: none"> Painful intermittent swelling of gland before/during meals Salivary colic due to obstruction of outflow of saliva 	<ul style="list-style-type: none"> Tenderness over affected gland Little/no saliva pours out from orifice of Wharton's duct on affected side Detection of stone on bidigital palpation or visible at opening of duct 	<ul style="list-style-type: none"> Radiography X-rays detects radioopaque stones in gland or duct Sialography or CT scan for radiolucent stones

Treatment

Operative

- Removal of stone in duct intraorally under local or general anaesthesia
 - Grasp tissue immediately behind stone with tenaculum forceps to steady and elevate stone
 - Incise long axis of duct letting stone to slip out
 - Leave wound unsutured.
- Sialadectomy
 - Removal of gland in case of stone in gland, recurrent stones or unpalpable stones.

22. Ludwig's angina.

Refer Question No. 5 December 2011 (RS2).

MBBS PHASE III EXAMINATION

DECEMBER 2017

(Revised Scheme 2 & 3)

■ LONG ESSAYS

1. Describe the etiology, clinical features and management of Meniere's disease.

Refer Question No. 2 December 2008 (RS2).

2. Describe etiopathogenesis, clinical features and management of ethmoidal polypsis.

Refer Question No. 2 December 2013 (RS2).

■ SHORT ESSAYS

3. Mucociliary blanket of nose.

- Mucociliary blanket is a continuous blanket of mucoserous secretion, secreted by goblet cells and secretory glands of nasal mucosa along with the cilia.

Components

- Superficial mucus layer
- Deeper thin serous layer, floating on the top of cilia.

Ciliary Movements (Physiology)

- Cilia under the mucus acts like conveyer belt, constantly beating towards the nasopharynx
- Movements of cilia have a rapid "effective stroke" and a slow "recovery stroke."
- In effective stroke, the extended cilia reach mucus layer while in recovery stroke, they bend and travel slowly in the reverse direction in the thin serous layer, thus moving mucus blanket in only one direction
- Cilia beat at a speed of 5–10 mm/min and every 10–20 min complete sheet of mucus is cleared into the pharynx
- To replenish this, about 600–700 mL of nasal secretions are produced in 24 hours.

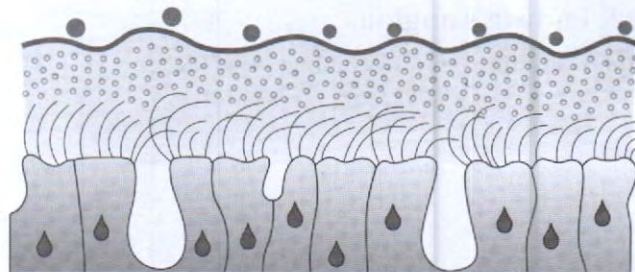


Figure 1: Mucociliary blanket of nose

Functions (Significance)

Protection

- Any inspired bacteria, viruses and dust particles are entrapped on viscous mucus blanket and then carried to the nasopharynx to be swallowed
- Nasal turbinates almost doubles the surface area of mucociliary blanket to perform this function.

Testing of Mucociliary Flow

- a. Indigo carmine test/saccharin sodium test
- A drop of indigo carmine (8 mg/ml) and a drop of saccharin sodium are put at anterior part of floor of inferior meatus

- After 3 minutes, patient is asked to swallow for 30 minutes and tell any taste of sweet
- Examiner simultaneously inspects posterior pharyngeal wall at these intervals for blue dye of indigo carmine
- Time lag between perception of sweet taste and appearance of blue dye in pharynx is noted
- This time lag is called mucus transport time (MTT).

Inference

- 12–15 minutes: Normal MTT (for ciliary beat frequency of 10/sec and transit time of 6 mm/min)
- >30 minutes: Grossly abnormal MTT.

Clinical Importance

- Kartagener's syndrome (triad of chronic rhinosinusitis, bronchiectasis and situs inversus) is due to immotile cilia syndrome, where cilia are defective (absence of dynein arm on periphery ciliary microtubules) and cannot beat effectively, leading to stagnation of mucus in the nose and sinuses and bronchi causing chronic rhinosinusitis and bronchiectasis
- Ciliary movements are affected by drying, drugs (adrenaline), excessive heat or cold, smoking, infections and noxious fumes like CO₂ and SO₂.

4. Juvenile laryngeal papillomatosis.

Refer Question No. 11 December 2007 (RS2).

5. Management of dysphagia.

Refer Question No. 9 December 2010 (RS2).

6. Nasal bone fractures.

Refer Question No. 3 December 2010 (RS2).

7. Ototoxicity.

Refer Question No. 5 December 2008 (RS2).

8. Preauricular sinus.

- Preauricular sinus is a common congenital anomaly of the ear
- It is a blind tract, lined by squamous epithelium.

Etiopathogenesis

- Due to faulty union of hillocks of 1st and 2nd branchial arches during development of pinna
- Commonly seen in between tragus and crux of helix (root of helix).

Clinical Features

- Sinus presents as an asymptomatic small opening in front of crus of helix (may be unilateral or bilateral)
- May get repeatedly infected (abscess formation) presenting with painful swelling and purulent discharge.

Treatment

- Abscess can be managed by antibiotics or incision and drainage
- Surgical excision of complete sinus tract to avoid recurrence (if there is unsightly swelling or infection).

Complication

Blocking of sinus results in retention preauricular cyst.

9. Fungal sinusitis.

Refer Question No. 8 December 2009 (RS2).

10. Clinical features and management of acute epiglottitis.

Refer Question No. 8 June 2008 (RS2).

11. Malignant otitis externa.

Refer Question No. 11 December 2010 (RS2).

12. Ludwig's angina.

Refer Question No. 5 December 2011 (RS2).

SHORT ANSWERS**13. Fistula test.**

Refer Question No. 3 Oct. 2009 (RS2).

14. Septal hematoma.

- Haematoma septum is collection of blood under perichondrium/periosteum of the nasal septum.

Etiology

- Blunt injury to nose as in road traffic accidents, boxing, etc
- Septal surgery (SMR, septoplasty)
- Bleeding disorders (spontaneous).

Clinical Features

Symptoms	Signs	Investigations
<ul style="list-style-type: none"> ♦ Bilateral constant and acute nasal obstruction ♦ Mouth breathing ♦ Frontal headache ♦ Sense of pressure over nasal bridge 	<ul style="list-style-type: none"> ♦ Smooth, biconvex swelling on either side of septum ♦ Soft, fluctuant mass (on palpation) 	<ul style="list-style-type: none"> ♦ Complete blood picture ♦ ESR ♦ Bleeding, clotting and prothrombin time ♦ X-ray paranasal sinuses

Treatment

Conservative	Operative	Complications
<ul style="list-style-type: none"> ♦ Systemic antibiotics and analgesics ♦ Anterior nasal packing or quilting sutures to prevent recurrence 	<ul style="list-style-type: none"> ♦ Aspiration with wide bore needle (small hematoma) ♦ Incision and drainage through small incision parallel to floor (large hematoma) 	<ul style="list-style-type: none"> ♦ Thickened septum (due to fibrosis in undrained hematoma) ♦ Septal abscess (due to secondary infection) ♦ Depression of nose (due to necrosis of cartilage)

15. Olfactory area.

Refer Question No. 3 June 2012 (RS2).

16. Laryngocele.

- Laryngocele is an air filled cystic swelling of larynx due to dilatation of saccule.

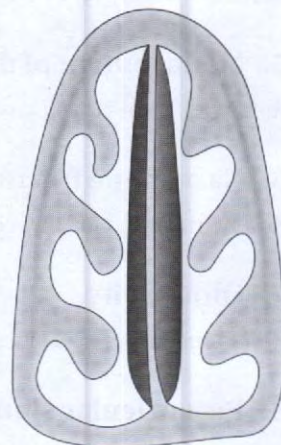


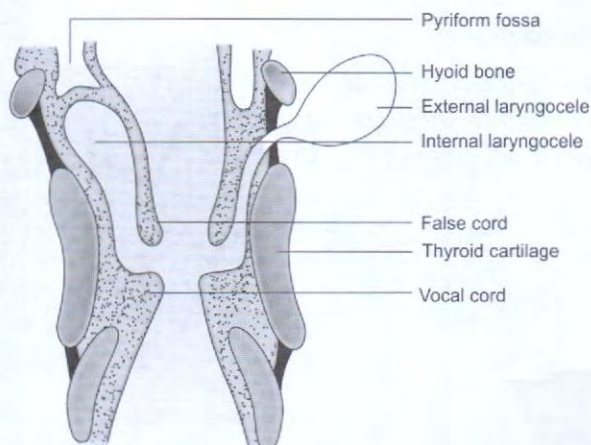
Figure 2: Septal hematoma

Etiology

- Congenital
- Acquired
 - Raised intraglottic air pressure
 - Chronic cough, asthma

Types

- Internal** Confined within larynx producing distension under vestibular bands and aryepiglottic folds
- External** Distended sacculus herniates through thyroid membrane and presents a compressible swelling in region of thyroid cartilage
- Combined or mixed** Both internal and external components

**Figure 3: Laryngocele – Types****Clinical Features**

- Seen in trumpet players, glass blowers or weight lifters
- Common in males

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Asymptomatic ♦ Hoarseness of voice ♦ Cough ♦ Dyspnea (due to pressure changes if large) 	<ul style="list-style-type: none"> ♦ Reducible swelling in neck which increases in size on coughing or performing Valsalva (external type) ♦ Gurgling and hissing sound in throat on external compression of neck swelling (Bryce's sign)

**Figure 4: Internal laryngocele**

Investigations	Treatment
<ol style="list-style-type: none"> Radiography <ul style="list-style-type: none"> – X-ray AP and lateral view of neck with Valsalva reveals air filled sac becoming prominent with Valsalva – CT helps to find extent of lesion Direct laryngoscopy <ul style="list-style-type: none"> – To rule out malignancy 	Operative <ul style="list-style-type: none"> ♦ Marsupialisation by laryngoscopy for internal laryngocele ♦ Surgical excision through transcervical incision

17. Leukoplakia of oral cavity.

Refer Question No. 13 December 2007 (RS2).

18. Tonsilloliths.

- Tonsilloliths (Tonsillolith) is presence of calculus (stone) in the crypts of tonsil
- Also called as Tonsillar concretions.

Etiopathogenesis

- Seen as complication of chronic tonsillitis when its crypt is blocked with retention of debris
- Inorganic salts of calcium and magnesium are then deposited leading to formation of a stone.

Clinical Features

- More often seen in adults
- Usually affects upper pole of tonsil.

<i>Symptoms</i>	<i>Signs</i>	<i>Diagnosis</i>
♦ Local discomfort or foreign body sensation	♦ Unilateral enlargement of affected tonsil ♦ Halitosis and sore throat (due to bacterial growth in retained material) ♦ Whitish foul-tasting and foul-smelling cheesy material expressed from tonsil	♦ Palpation or gritty feeling on probing

Treatment

<i>Conservative</i>	<i>Operative</i>
♦ Expression of concretions/cheesy material and chemical cauterization of crypts with topical silver nitrate application	♦ Simple removal of the stone ♦ Tonsillectomy (if associated persistent pain, sepsis or deeply set stone)

Complications

- Stone may gradually enlarge and then ulcerate on medial surface of tonsil.

19. Glue ear.

Refer Question No. 5 December 2013 (RS2).

20. Ramsay Hunt syndrome.

Refer Question No. 7 June 2009 (RS2).

21. Blood supply of nasal septum.

Refer Question No. 7 June 2017 (RS2).

22. Vocal nodule.

Refer Question No. 9 June 2016 (RS2).

SAMPLE PAPERS

MBBS PHASE III EXAMINATION

SAMPLE PAPER-1

LONG ESSAYS

1. Enumerate the causes of vocal cord paralysis. How do you investigate and manage a case of unilateral vocal cord paralysis?

- Injury to nerves supplying vocal cords results in vocal cord paralysis.

Causes of Vocal Cord Paralysis

- Supranuclear: Bilateral
 - Rare as larynx has bilateral representation in motor cortex
- Nuclear (involvement of nucleus ambiguus in medulla): Bilateral
 - Vascular causes such as thrombosis or hemorrhage of posterior cerebellar artery
 - Tumors
 - Motor neuron diseases
 - Bulbar poliomyelitis, diphtheria
 - Syringobulbia
 - Disseminated sclerosis
- High vagal lesions: Unilateral or bilateral
(Combined paralysis of superior and recurrent laryngeal nerves)

<i>Intracranial</i>	<i>Skull base</i>	<i>Neck</i>
<ul style="list-style-type: none"> ♦ Stroke ♦ Tumors of posterior fossa ♦ Basal meningitis (acute or chronic) ♦ Meningomyelocele ♦ Tuberculoma ♦ Head injury 	<ul style="list-style-type: none"> ♦ Basal fractures ♦ Nasopharyngeal cancer ♦ Glomus tumor ♦ Osteomyelitis of base of skull 	<ul style="list-style-type: none"> ♦ Penetrating injury ♦ Parapharyngeal tumors ♦ Metastatic nodes ♦ Lymphoma ♦ Glomus vagale ♦ Neurilemmoma of vagus nerve ♦ Extracalvarial meningioma

- Low vagal lesions: Unilateral or bilateral
(Paralysis of recurrent laryngeal nerves)

<i>Right recurrent laryngeal nerve—unilateral</i>	<i>Left recurrent laryngeal nerve—unilateral</i>	<i>Both recurrent laryngeal nerve—bilateral</i>
<i>In Neck</i>		
<ul style="list-style-type: none"> ♦ Neck trauma ♦ Benign or malignant neoplasm of thyroid ♦ Thyroid surgery ♦ Cervical esophageal cancer ♦ Cervical lymphadenopathy 	<ul style="list-style-type: none"> ♦ Neck trauma ♦ Benign or malignant neoplasm of thyroid ♦ Thyroid surgery ♦ Cervical esophageal cancer ♦ Cervical lymphadenopathy 	<ul style="list-style-type: none"> ♦ Thyroid carcinoma ♦ Thyroid surgery ♦ Cervical esophageal cancer ♦ Cervical lymphadenopathy

<i>Right recurrent laryngeal nerve—unilateral</i>	<i>Left recurrent laryngeal nerve—unilateral</i>	<i>Both recurrent laryngeal nerve—bilateral</i>
<i>In mediastinum</i>		
<ul style="list-style-type: none"> ♦ Idiopathic ♦ Aneurysm of subclavian artery ♦ Carcinoma of apex of right lung ♦ Tuberculosis of cervical pleura 	<ul style="list-style-type: none"> ♦ Idiopathic ♦ Aortic aneurysm ♦ Enlarge left auricle with mitral stenosis (Ortner's syndrome) ♦ Tuberculosis of lungs ♦ Bronchogenic carcinoma ♦ Thoracic esophageal carcinoma ♦ Mediastinal lymphadenopathy ♦ Intrathoracic operations 	

e. Systemic causes: Bilateral

- Diabetes
- Syphilis
- Diphtheria
- Typhoid
- Streptococcal infection
- Viral infection
- Lead poisoning

f. Idiopathic (30%): Unilateral or bilateral.

Unilateral Vocal Cord Paralysis

- Unilateral vocal cord paralysis is involvement of one side of vocal cords either left or right.

Classification

- Recurrent laryngeal nerve palsy
 - Incomplete (only abductors paralyzed)
 - Complete (both abductors and adductors paralyzed)
- Superior laryngeal nerve palsy
- Combined paralysis.

Clinical Features

- Usually affects adults
- Both sexes equally affected.

	<i>Incomplete recurrent laryngeal nerve palsy</i>	<i>Complete recurrent laryngeal nerve palsy</i>	<i>Superior laryngeal nerve palsy</i>	<i>Combined palsy</i>
Symptoms				
♦ Voice	♦ Normal	♦ Hoarseness	♦ Weak, monotonous	♦ Hoarseness and feeble, rough voice
♦ Respiration	♦ Normal	♦ Normal	♦ Normal	♦ Normal
♦ Swallowing	♦ Normal	♦ Normal	♦ Inhalation into larynx (due to sensory paralysis)	♦ Inhalation into larynx (due to sensory paralysis)
♦ Signs (indirect laryngoscopy)	♦ Vocal cords in median ♦ Shallow pyriform fossa ♦ Forward fall of arytenoids ♦ Paralyzed cord at lower level ♦ Leaning forward of cartilage of Wrisberg	♦ Vocal cords in paramedian position ♦ Shallow pyriform fossa ♦ Forward fall of arytenoids ♦ Paralyzed cord at lower level ♦ Leaning forward of cartilage of Wrisberg	♦ Oblique laryngeal inlet ♦ Bowing of affected cord during phonation ♦ Bulky cords ♦ Deviation of posterior commissure towards paralyzed side	♦ Vocal cords in cadaveric position ♦ Wavy and lax vocal cords without tension

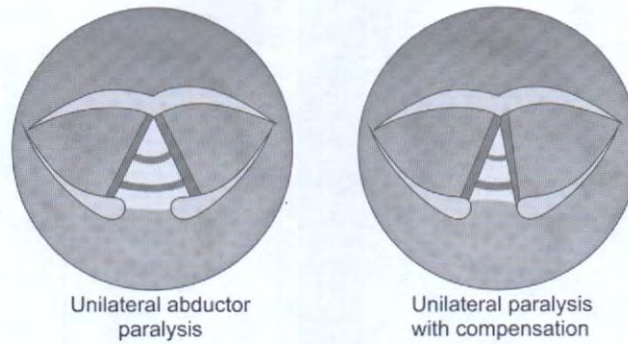


Figure 1: Unilateral vocal cord paralysis

Investigations

a. Blood investigations

- Erythrocyte sedimentation rate (ESR)
- Blood sugar
- Venereal disease research laboratory (VDRL)
- Raised in malignancy, tuberculosis
- To detect diabetes
- To diagnose syphilis

b. Radiological investigations

- X-ray chest (PA and lateral view)
- Barium swallow
- Computed tomography (CT) scan and magnetic resonance imaging (MRI)
- May reveal mediastinal mass like aneurysm of aorta, pulmonary tuberculosis, enlarged heart, etc
- May detect carcinoma of esophagus
- To detect any space occupying lesion in cranium or thorax

c. Endoscopies

- Direct laryngoscopy
- Bronchoscopy and esophagoscopy
- To detect occult primaries in anterior commissure of larynx or subglottic growth
- To detect any malignant growth

d. Neurological investigations

- CT scan, MRI, carotid angiograms and fundoscopy
- For intracranial lesions

e. Cardiovascular investigations

- Echocardiography, angiographs
- For aortic aneurysm, mitral stenosis, etc.

Treatment

Asymptomatic cases (incomplete recurrent laryngeal nerve palsy)

- No treatment

Symptomatic cases (other palsies)

Supportive

- Treatment of hoarseness

Conservative

a. Speech therapy

- Helps to compensate loss of function of paralyzed cord by moving of healthy cord across midline

Operative (if paralysis persists >9–12 months)

a. Injection of Teflon paste

- Teflon glycerine paste is injected lateral to paralyzed vocal cords to push paralyzed cord medially

b. Implantation of cartilage or muscle

- Bipedicled muscle graft or pieces of cartilage is inserted between thyroid cartilage and its inner perichondrium lateral to paralyzed vocal cord thus pushing cord medially

c. Arthrodesis of cricoarytenoid joint

- Rotate arytenoid cartilage medially and fix with a screw

- d. Thyroplasty type I (phonosurgery)
– Paralyzed cord medialized by silastic implants.

Specific

- Treatment of underlying cause.

2. Describe the causes and management of acute earache.

- Pain in ear (earache) is called "Otalgia".

Etiology

A. Local Causes

External ear (TOWN OF Finance Minister)	Middle ear (BECAME)
<ul style="list-style-type: none"> ♦ Trauma ♦ Otitis externa ♦ Wax (commonest cause) ♦ Neoplasms ♦ Otomycosis ♦ Furuncle ♦ Foreign body ♦ Myringitis bullosa 	<ul style="list-style-type: none"> ♦ Barotrauma ♦ Eustachian tube obstruction (commonest cause) ♦ Carcinoma of middle ear ♦ Acute otitis media and its complications ♦ Mastoiditis ♦ Extradural abscess

B. Referred Causes

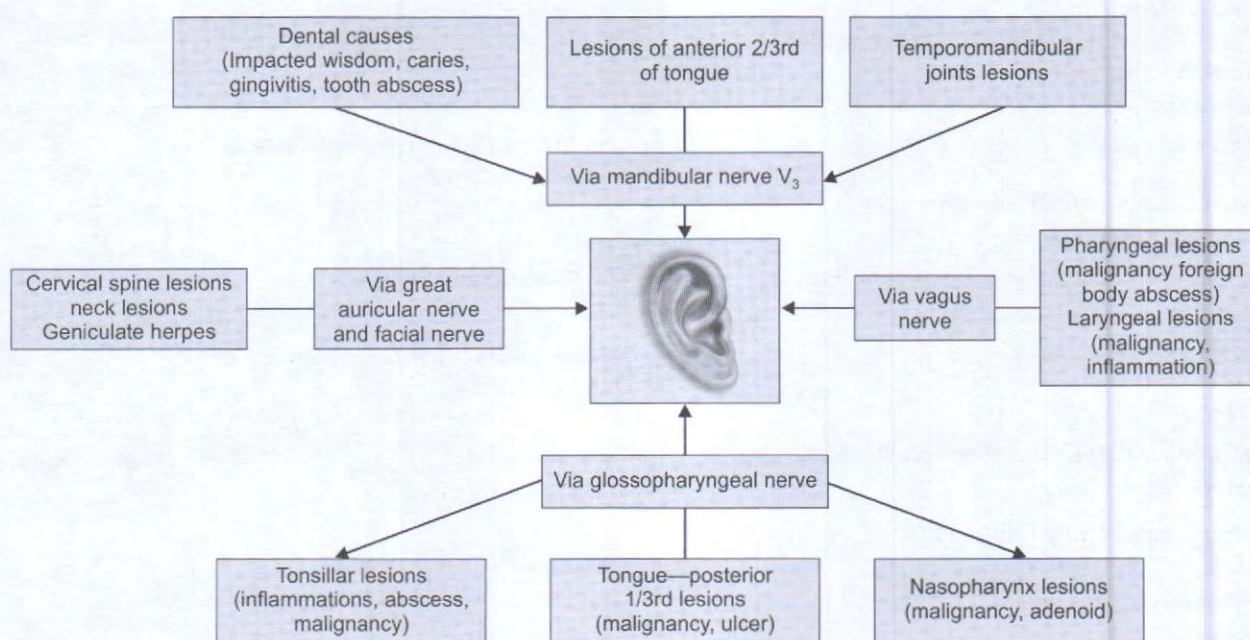


Figure 2: Causes of referred earache

a. Via trigeminal nerve (V nerve): Auriculotemporal branch:

Dental (Common AIM)	Oral cavity (SUI)	TM joint	Nose, paranasal sinuses (DIM)	Nasopharynx (CAN)	Sphenopalatine neuralgia
<ul style="list-style-type: none"> ♦ Caries ♦ Alveolar abscess ♦ Impacted molar ♦ Malocclusion 	<ul style="list-style-type: none"> ♦ Salivary calculus ♦ Ulcerative lesions (benign/malignant) ♦ Infection 	<ul style="list-style-type: none"> ♦ Arthritis ♦ Bruxism 	<ul style="list-style-type: none"> ♦ Deviated nasal septum ♦ Infection (Sinusitis) ♦ Malignancy 	<ul style="list-style-type: none"> ♦ Carcinoma ♦ Adenoidectomy ♦ Nasopharyngitis 	

- b. Via facial nerve (VIII nerve)—greater auricular nerve
- Herpes zoster
 - Cervical spine lesions
 - Neck lesions
- c. Via glossopharyngeal nerve (IX nerve)

<i>Tonsils</i>	<i>Oropharynx</i>	<i>Elongated styloid process</i>	<i>Neuralgia</i>
<ul style="list-style-type: none"> ♦ Acute tonsillitis ♦ Quinsy ♦ Post-tonsillectomy pain ♦ Malignancy 	<ul style="list-style-type: none"> ♦ Infection (retropharyngeal and parapharyngeal abscess) ♦ Trauma ♦ Malignancy 		

- d. Via vagus nerve (X nerve)
- Vallecula, epiglottis, larynx, esophagus, laryngopharynx
 - Trauma
 - Ulcerative lesions
 - Tuberculosis
 - Malignancy.
- e. Via C2 and C3 spinal nerves
- Cervical spondylolysis
 - Cervical spine
 - Fibrositis
 - Myositis
 - Trauma to cervical spine.
- C. Psychogenic
- Non-organic pain.

Clinical Features

- Pain in ear which increases on lying down (due to increased blood supply)
- Features of underlying cause.

Treatment

<i>Supportive</i>	<i>Specific</i>
<ul style="list-style-type: none"> ♦ Analgesics to relieve pain ♦ Anesthetic ear droop to sooth earache 	<ul style="list-style-type: none"> ♦ Treatment of underlying cause

SHORT ESSAYS

3. Role of cochlea in hearing.

- Cochlea is a component of inner ear housing end organ of hearing, i. e. organ of Corti.

Role of Cochlea in Hearing

- Cochlear components namely basilar membrane, hair cells and tectorial membrane act in integrated fashion to converts mechanical form of energy into appropriate action potential for precise perception of sound.

Mechanism

- Scala vestibuli and scala tympani of cochlea are filled with perilymph
- Scala vestibuli and scala tympani are continuous with each other at apex of cochlea and end in oval window and round window respectively

- Cochlea contains end organ of hearing called organ of Corti, spread along entire basilar membrane, enclosed in tunnel of Corti filled with cortilymph
- Organ of Corti contains hair cells, axons of which continues as cochlear nerve
- When sound from tympanic membrane is transmitted to oval window through ossicular chain, there occurs transmission of this mechanical energy from foot plate of stapes into vibration of perilymph filled in scala vestibuli
- Vibration of perilymph sets basilar membrane in motion producing what is called traveling wave which travels along basilar membrane
- Basilar membrane exhibit variable response to variable frequencies along its length, i.e. it responds to higher frequencies at beginning and gradually receding to lower frequencies towards apex
- Depending upon frequency of sound, there occurs maximum bowing of basilar membrane at certain point corresponding to that frequency
- Bowing of basilar membrane causes movement of cilia of hair cells eventually resulting in generation of action potential which is transmitted through cochlear nerve to auditory center for perception of sound.

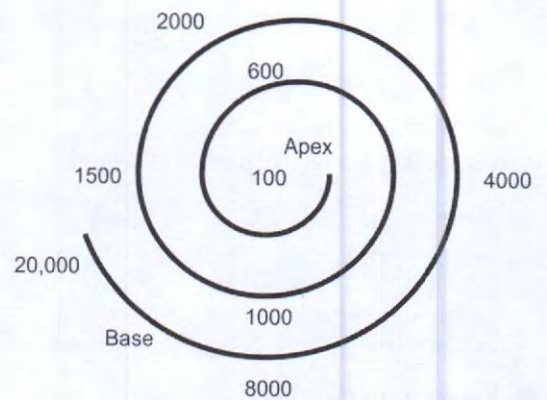


Figure 3: Cochlea in hearing

Causes of vertigo—VERTIGO

- V-Vascular
- E-Eustachian catarrh
- R-Remedies (ototoxic drugs)
- T-Trauma, Tumors
- I-Infections
- G-Glomus tumor
- O-Other (Meniere's disease, motion sickness)

Significance

- Cochlea acts as a fine transducer and frequency analyzer of sound in terms of pitch discrimination
- It encodes frequency and amplitude of sound waves that is further processed in auditory pathway higher up in cortex.

4. Causes of Vertigo.

- Vertigo is a disturbance of sense of equilibrium and movements.

Aural Causes of Vertigo

A. External ear	
<ul style="list-style-type: none"> - Wax - Furuncle 	- Due to stimulation of auricular branch of vagus nerve—rarely
B. Middle ear	
<ul style="list-style-type: none"> - Eustachian catarrh - Otitis media 	<ul style="list-style-type: none"> - Negative pressure in middle ear may be transmitted to inner ear causing giddiness - Acute otitis media and chronic dangerous otitis media cause giddiness by producing labyrinthitis
C. Inner ear	
<ul style="list-style-type: none"> - Trauma 	<ul style="list-style-type: none"> - May cause giddiness along with sensorineural deafness
<ul style="list-style-type: none"> - Head injuries with longitudinal fracture of temporal bone involvement of labyrinth - Surgical trauma - Acoustic trauma - Perilymph fistula 	<ul style="list-style-type: none"> - Vestibule may be damaged during mastoidectomy or stapedectomy - Very loud sound may occasionally cause vertigo (Tullio phenomenon) - Barotrauma may cause rupture of round window membrane resulting in perilymph fistula - It may also result during stapedectomy, ear surgery - May cause concussion of labyrinth
<ul style="list-style-type: none"> - Whiplash injury 	
<ul style="list-style-type: none"> - Infections 	
<ul style="list-style-type: none"> - Labyrinthitis 	<ul style="list-style-type: none"> - Secondary to acute otitis media, dangerous chronic otitis media with cholesteatoma, tuberculous or syphilitic otitis media

	<ul style="list-style-type: none"> - Secondary to meningitis - Due to mumps, measles, herpes zoster - Syphilis
- Vascular causes	- Thrombosis, embolism of blood vessels supplying labyrinth
- Tumors	<ul style="list-style-type: none"> - Acoustic neuroma (neurofibroma arising from VIII nerve in internal auditory meatus) - Tumors of temporal bone (Glomus tumor, carcinoma of external or middle ear)
- Meniere's syndrome (group of otological diseases with vertigo as dominating syndrome)	<ul style="list-style-type: none"> - Meniere's disease - Vestibular neuronitis - Benign paroxysmal positional vertigo - Lermoyez syndrome - Acoustic neuroma
- Motion sickness	- Due to hypersensitive labyrinth, with functional overlay due to fear of recurrence
- Ototoxic drug	<ul style="list-style-type: none"> - Aminoglycosides - Labyrinthine sedatives - Antihypertensives - Diuretics

<i>Clinical features</i>	<i>Investigations</i>	<i>Treatment</i>
Symptoms <ul style="list-style-type: none"> ♦ Sense of mild imbalance or dizziness (feeling of surrounding going round him or feeling of himself rotating) ♦ Nausea and vomiting (in severe vertigo) ♦ Perspiration, gastric upset and diarrhea (due to vagal stimulation) Signs <ul style="list-style-type: none"> ♦ Evidence of underlying etiology 	<ul style="list-style-type: none"> ♦ Tests of hearing (Tuning fork tests) ♦ Labyrinthine tests (Caloric test, Electronystagmography) ♦ Pathological tests (VDRL test) ♦ Radiological examination (X-ray mastoid and skull, CT scan, MRI) ♦ Neurological examination (cerebral angiography) 	Supportive <ul style="list-style-type: none"> ♦ Reassurance ♦ Labyrinthine sedatives (prochlorperazine) ♦ Vasodilators (nicotinic acid) Specific <ul style="list-style-type: none"> ♦ Treatment of underlying cause

5. Cauliflower ear.

- Cauliflower ear is a condition of deformed pinna occurring as complication of perichondritis or hematoma of ear
- Also called Pugilistic or Boxer's ear

<i>Etiopathogenesis</i>	<i>Clinical feature</i>
Recurrent trauma to ear as in boxers ↓ Hematoma ↓ Organization of hematoma ↓ Cauliflower ear	<ul style="list-style-type: none"> ♦ Seen commonly in Boxer's, wrestlers and rugby players who are subjected to recurrent trauma to ear Symptoms <ul style="list-style-type: none"> ♦ Deformed pinna ♦ Feeling of burning sensation and warmth in ear ♦ Stiff and painful movement of pinna ♦ Diffuse/multiple cystic swellings ♦ Fever, bodyache Signs <ul style="list-style-type: none"> ♦ Tenderness

Treatment

<i>Conservative</i>		<i>Operative</i>
<i>Supportive</i>	<i>Specific</i>	
<ul style="list-style-type: none"> ♦ Local application of aluminum acetate ♦ Pressure bandage ♦ Application of 20% silver nitrate or phenol for granulation tissue cautery ♦ NSAIDs to relieve pain 	<ul style="list-style-type: none"> ♦ Prompt and complete treatment of perichondritis and hematoma auris with antibiotics 	<ul style="list-style-type: none"> ♦ Incision and drainage of hematoma under strict aseptic precautions

6. Draw neat labeled diagrams showing different types of perforations of the tympanic membrane.

- Tympanic membrane may be perforated due to trauma or diseases.

Types (Based on Location)

<i>Pars tensa</i>		<i>Pars flaccid</i>
Central perforation (annulus preserved)	Marginal perforation (annulus destroyed and reaches sulcus tympanicus)	
<ul style="list-style-type: none"> ♦ Anterior (anterior to handle of malleus) ♦ Posterior (posterior to handle of malleus) ♦ Inferior (inferior to handle of malleus) ♦ Subtotal (very large perforation) 	<ul style="list-style-type: none"> ♦ Posterosuperior ♦ Anterior ♦ Inferior ♦ Total 	<ul style="list-style-type: none"> ♦ Attic perforation

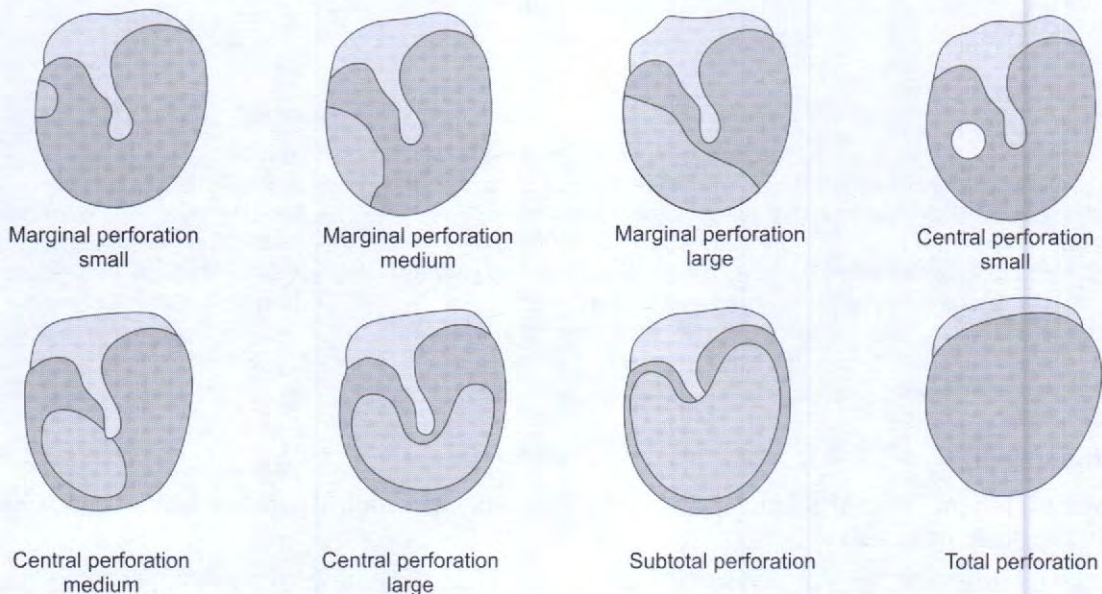


Figure 4: Tympanic membrane perforations

Clinical Significance

- Attic and posterosuperior marginal perforations are seen in dangerous type of CSOM and often associated with cholesteatoma
- Marginal perforations shows ingrowth of stratified squamous epithelium from external auditory canal to form cholesteatoma (thus making them dangerous)
- Central perforations are not associated with cholesteatoma (hence considered safe).

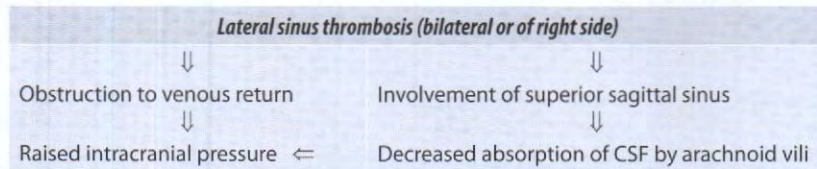
7. Otitic hydrocephalus.

- Otitic hydrocephalus is an intracranial complication of suppurative otitis media characterized by raised intracranial pressure with normal [cerebrospinal fluid (CSF)] findings.

Etiology

- Lateral sinus thrombosis.

Pathogenesis



Clinical Features

- Common in children and adolescents
- Gradual onset

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Severe headache <ul style="list-style-type: none"> – Intermittent – Accompanied by nausea and vomiting ♦ Visual disturbances <ul style="list-style-type: none"> – Blurring of vision due to papilledema or optic atrophy – Diplopia due to paralysis of 6th cranial nerve 	<ul style="list-style-type: none"> ♦ Papilledema <ul style="list-style-type: none"> – Optic disc edema of 5–6 diopters – Patches of exudates and hemorrhages in retina ♦ Nystagmus

Investigation	Treatment	
	Conservative	Operative
<ul style="list-style-type: none"> ♦ Diagnostic lumbar puncture <ul style="list-style-type: none"> – CSF pressure >300 mm of water ♦ CSF examination <ul style="list-style-type: none"> – Normal and sterile 	<ul style="list-style-type: none"> ♦ Diuretics like acetazolamide ♦ Corticosteroids ♦ Antibiotics to treat middle ear infection 	<ul style="list-style-type: none"> ♦ Lumbar puncture and drain ♦ Lumboperitoneal shunt

8. Discuss nasal foreign body.

- Nasal foreign body is an usual presenting complaint in children coming to ENT OPD.

Etiology

- Accidental (in children)
- Iatrogenic (postoperative).

Classification

Non living		Living	Iatrogenic
Organic	Inorganic		
<ul style="list-style-type: none"> ♦ Seeds ♦ Grains and grams 	<ul style="list-style-type: none"> ♦ Piece of paper ♦ Chalk ♦ Button ♦ Pebbles 	<ul style="list-style-type: none"> ♦ Maggots 	<ul style="list-style-type: none"> ♦ Cotton or swabs

Clinical Features

- Common in children

Symptoms		Signs
Immediately	Neglected cases	
<ul style="list-style-type: none"> ♦ History of foreign body insertion <ul style="list-style-type: none"> – Sneezing due to irritation – Bleeding from nose – Blocking sensation – Pain 	<ul style="list-style-type: none"> ♦ Nasal discharge <ul style="list-style-type: none"> – Unilateral – Often foul smelling and occasionally blood stained (characteristic feature) 	<ul style="list-style-type: none"> ♦ Dermatitis of upper lip below affected nostril ♦ Foreign body seen on anterior rhinoscopy ♦ Sometimes may be hidden behind discharge and granulations

Investigations

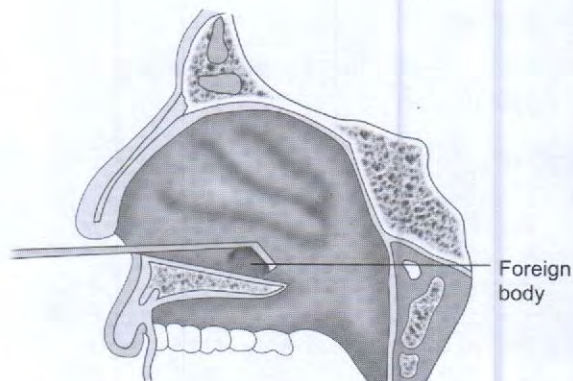
- ♦ Radiograph
 - Radio-opaque foreign bodies may be detected on X-ray nose
- ♦ Diagnostic nasal endoscopy
 - Helps in thorough inspection of nasal cavity and visualization of clinically nonvisible foreign bodies

Differential diagnosis

- ♦ Diphtheria
 - Bilateral
 - Excoriation spreading beyond nasal cavity to external nose
- ♦ Rhinolith and sequestra
 - Presents as hard irregular masses

Treatment

- a. First aid
 - Blowing of nose or induction of sneezing may expel foreign body
- b. Removal
 - Removal of foreign body using instruments either through anterior nares or nasopharynx (by pushing it backward)
 - Forceps for pieces of paper, cotton swabs, etc.
 - Blunt hook for rounded foreign bodies
 - General anesthesia needed in uncooperative patients or for large foreign bodies.

**Figure 5:** Removal of nasal foreign body**Complications**

- Nasal infection and sinusitis
- Rhinolith formation
- Inhalation into tracheobronchial tree.

9. Unilateral nasal obstruction—Causes.

- Unilateral nasal obstruction is an important differential diagnosis for antrochoanal polyp.

Differential Diagnosis (Causes)

Vestibule	Nasal cavity	Nasopharynx
<ul style="list-style-type: none"> ♦ Furuncle ♦ Vestibulitis ♦ Stenosis of nares ♦ Atresia ♦ Nasoalveolar cyst ♦ Papilloma ♦ Squamous cell carcinoma 	<ul style="list-style-type: none"> ♦ Foreign body ♦ Deviated nasal septum ♦ Hypertrophic turbinates ♦ Concha bullosa ♦ Antrochoanal polyp ♦ Synechia ♦ Rhinolith ♦ Bleeding polyposus of septum ♦ Tumors of nose and paranasal sinuses ♦ Sinusitis (unilateral) 	<ul style="list-style-type: none"> ♦ Choanal atresia (unilateral)

10. Vincent's angina.

- An acute ulcerative lesion of pharynx involving one or both tonsils
- Also called trench mouth, acute membranous pharyngitis or tonsillitis.

Etiology

Causative organisms	Predisposing factors
<ul style="list-style-type: none"> ♦ Fusiform bacillus (Vincent's organism) ♦ Spirochete denticola 	<ul style="list-style-type: none"> ♦ Poor dental hygiene ♦ Caries teeth ♦ Pyorrhea ♦ Overcrowding ♦ Poor diet ♦ General debility

Clinical Features

- Usually affects young adults and middle aged people
- Sudden onset.

Symptoms	Signs
<ul style="list-style-type: none"> ♦ High grade fever ♦ Severe pain in throat ♦ Bad smell from mouth (fishy odor) 	<ul style="list-style-type: none"> ♦ Affected area covered with greyish membranous slough which on removal causes considerable loss of tissue leaving behind bleeding base ♦ Slough reforms after removal ♦ Cervical lymphadenitis

Investigations

- Pharyngeal swab
 - Gram staining and culture
 - Hanging drop preparation.

Treatment

Conservative	Operative
<ul style="list-style-type: none"> ♦ Systemic broad spectrum antibiotics (metronidazole and clindamycin) ♦ Analgesics to relieve pain and fever ♦ Antiseptic mouth wash ♦ Antiseptic paint over fauces and gum 	<ul style="list-style-type: none"> ♦ Surgical debridement

11. Tuberculous cervical lymphadenitis.

- Tuberculous lymphadenitis is chronic specific infection of lymph nodes
- Commonly affects lymph nodes of neck.

Causative organism	Mode of infection	Stages
♦ Mycobacterium tuberculosis	<ul style="list-style-type: none"> ♦ From tonsils—usually ♦ From blood from lungs—occasionally ♦ From tuberculous lesion of apex of lungs through suprapleural fascia—rarely 	<ul style="list-style-type: none"> ♦ Stage of infection and lymphadenitis ♦ Stage of periadenitis with matting ♦ Stage of caseating necrosis with cold abscess formation ♦ Stage of formation of collar stud abscess ♦ Stage of formation of sinus discharging yellowish caseating material

Pathology

Gross appearance	Microscopic examination
<ul style="list-style-type: none"> ♦ Firm, matted lymph nodes ♦ Cut section shows yellowish caseating material 	<ul style="list-style-type: none"> ♦ Epithelioid cells with caseating material along with Langhan's type of giant cells

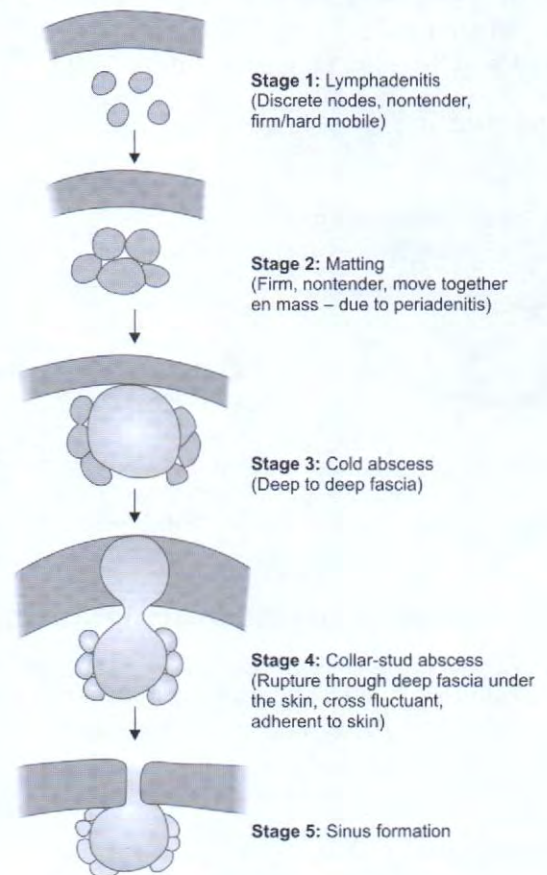


Figure 6: Stages of tuberculous lymphadenitis

Clinical Features

- Usually affects upper deep cervical lymph nodes (54%) followed by posterior triangle lymph nodes (22%).

Symptoms	Signs
Firm swelling in lateral side of neck	<ul style="list-style-type: none"> ♦ Firm and matted swelling in neck ♦ Cold abscess is soft, smooth, nontender, fluctuant without involvement of skin ♦ Rupture of cold abscess into deep fascia forms collar stud abscess which is adherent to overlying skin ♦ Bursting of abscess leads to multiple, wide mouthed, undermined, non-mobile discharging sinuses with bluish edge

Differential Diagnosis

- Nonspecific lymphadenitis
- Lymphomas and chronic lymphatic leukemia
- Secondaries in neck
- HIV with lymph node involvement
- Branchial cyst (mimics cold abscess)
- Lymph cyst (mimics cold abscess)
- Actinomycosis (mimics discharging sinus).

Investigations

- Erythrocyte sedimentation rate (ESR)
- Fine needle aspiration cytology (FNAC) or biopsy of lymph node (for culture and smear for acid fast bacilli)
- HIV test
- Chest X-ray (for pulmonary tuberculosis).

Treatment*Conservative*

- Antituberculous regime
 - According to Revised National Tuberculosis Control Program.

Operative

	Aspiration	Incision and drainage	Surgical removal
Indications	♦ Cold abscess	♦ Recurrence after aspiration	<ul style="list-style-type: none"> ♦ Failure of drug therapy ♦ Persistent sinus
Technique	♦ Using wide bore needle in nondependent site along Z track (Zig-zag pathway)	♦ Incise on nondependent location and after drainage of caseating material, close without placing a drain	♦ Remove all caseating material and lymph node along with excision of sinus track after raising skin flap

12. Write a note on microlaryngeal surgery.

- Microlaryngeal surgery is a precise operative procedure on larynx performed under operating microscope for a sufficient length of time during microlaryngoscopy
- Microlaryngoscopy is technique of visualization of larynx using laryngoscope fitted with operating microscope (with 400 mm objective)
- It is a refinement of direct laryngoscopy.

Requirement

- Self-retaining laryngoscope with chest piece
- Operating microscope with objective lens of 400 mm focal length.

Indications		Contraindications
Diagnostic	Therapeutic	
<ul style="list-style-type: none"> ♦ <u>Diagnosis and evaluation</u> of laryngeal diseases ♦ <u>Supravital staining</u> of vocal cords ♦ <u>Photography and videography</u> of endolarynx ♦ <u>Tissue biopsy</u> ♦ <u>Early detection of laryngeal cancers</u> and their extent 	<ul style="list-style-type: none"> ♦ <u>Removal of benign lesions</u> of larynx (cordal nodules, vocal polyps, cysts) ♦ <u>Foreign body removal</u> from larynx and hypopharynx ♦ <u>Removal of thickened secretions</u> and crust ♦ <u>Injection of telfon paste</u> in vocal cord for vocal cord palsy ♦ <u>Dilatation of laryngeal strictures</u> ♦ <u>Excision of premalignant lesions</u> like leukoplakia, carcinoma in situ ♦ <u>Endoscopic arytenoidectomy</u> in bilateral abductor palsy ♦ <u>Laser or cryosurgery</u> for laryngeal lesions 	<ul style="list-style-type: none"> ♦ <u>Cervical spine pathology</u> ♦ <u>Trismus</u> ♦ <u>Long incisor teeth</u> ♦ <u>Laryngeal spasms and stridor</u> ♦ <u>Moderate or marked dyspnea</u> ♦ <u>Short and thick neck</u> ♦ <u>Vascular tumors</u> like aneurysms of aorta ♦ <u>Recent coronary occlusion</u> or cardiac decompensation ♦ <u>Respiratory and cardiac diseases</u> (may cause extrasystole, cardiac arrest) ♦ <u>General contraindications</u> (anemia, diabetes, hypertension)

Anesthesia

- ♦ General anesthesia with endotracheal intubation using small cuffed tube.

Position

- ♦ Supine position with elevated head (10–15 cm) by placing pillow under occiput thus flexing neck at thorax and extending head at atlanto-occipital joint (Boyce's position or Barking dog position).

Procedure

- ♦ Place a piece of gauze on upper teeth to prevent injury to them
- ♦ Lubricate laryngoscope with autoclaved liquid paraffin or jelly
- ♦ Use left hand to hold handle of laryngoscope while right hand retracts lips and guides it into larynx and also handles suction and instruments
- ♦ Introduce laryngoscope into oral cavity through right side of angle of mouth pushing tongue to opposite side till base of tongue is reached
- ♦ Lift laryngoscope forwards and move it in midline to bring epiglottis in view
- ♦ Guide laryngoscope behind epiglottis and lift laryngoscope forward (do not lever it on upper teeth or jaw)—engagement of epiglottis
- ♦ Advance tip of laryngoscope for 1cm between ventricular bands (false vocal cords) to examine ventricles and anterior commissure
- ♦ Observe mobility of vocal cords
- ♦ Then pass is between vocal cords to examine subglottic region
- ♦ Undersurface of vocal cords and walls of subglottic can be visualized using right angled telescope
- ♦ Fix self-retaining laryngoscope to chest piece
- ♦ Perform operative procedure
- ♦ Remove laryngoscope in reverse fashion.

Complications	Advantages	Disadvantages
<ul style="list-style-type: none"> ♦ <u>Injury</u> to lips, tongue, teeth and vocal cords ♦ Trauma to larynx on repeated attempts ♦ <u>Bleeding</u> from operative site ♦ <u>Laryngeal edema</u> particularly of subglottic ♦ <u>Hypoxia and cardiac arrest</u> ♦ Dislocation of cervical vertebrae (if diseased) ♦ Slipping of foreign body or disintegration of foreign body 	<ul style="list-style-type: none"> ♦ <u>Direct, complete and true visualization</u> of larynx with magnification and excellent illumination ♦ <u>Precise and accurate surgery</u> ♦ <u>Photography or videography possible</u> for maintaining records ♦ Teaching possible by attaching camera to laryngoscope ♦ Can be coupled with laser surgery, micro-dissection ♦ Minimal damage to vocal cords 	<ul style="list-style-type: none"> ♦ <u>Invasive procedure</u> ♦ <u>Requires anesthesia</u> ♦ Needs to be done in <u>operation theater</u> ♦ <u>Costly instruments</u>

■ SHORT ANSWERS

13. Glottic cancers have high cure rate. Give reasons.

- Glottic cancer indicates cancer of larynx extending from floor of ventricle to about 10 mm inferior to free border of true cords
- It accounts for 50–75% of laryngeal cancers
- Early treatment of glottic cancer has high cure rate (90–95%) because
 - It presents early with hoarseness of voice
 - It rarely metastasizes lymphatically due to poor lymphatic drainage
- Hence glottic cancer has high cure rate if diagnosed and treated early.

14. Cerumen (Wax).

- Wax is collection of natural secretions of sebaceous glands, ceruminous glands, hair, desquamated epithelial debris, keratin and dirt.

Formation	Expulsion	Functions
<ul style="list-style-type: none"> ♦ Due to mixing of fatty acid rich fluid from sebaceous gland and lipid and pigment granule containing secretions of ceruminous glands with desquamated epithelium and keratin shed from tympanic membrane and deep bony meatus 	<ul style="list-style-type: none"> ♦ By movements of jaw 	<ul style="list-style-type: none"> ♦ Lubrication of ear canal ♦ Antibacterial action ♦ Trapping of foreign material entering ear canal

Pathology

Impaction of Wax

- Wax may get accumulated and harden leading to impaction.

Etiology	Clinical Features
<ul style="list-style-type: none"> ♦ Change in chemical composition of wax ♦ Excessive secretions by sebaceous or ceruminous glands ♦ Narrow and tortuous ear canal ♦ Stiff hairs ♦ Obstructive lesion of ear canal like exostosis ♦ Self-cleaning of ear (removing wax from outer ear while pushing wax in deeper part more medially) ♦ Dusty occupations ♦ Hot and dry climate 	<ul style="list-style-type: none"> ♦ Sudden onset of symptoms after exposure to water (as in swimming or bathing due to swelling of wax) <p><i>Symptoms</i></p> <ul style="list-style-type: none"> ♦ Pain ♦ Impaired hearing ♦ Sensation of blocked ear ♦ Itching ♦ Tinnitus and giddiness (wax impacted against tympanic membrane) ♦ Reflex cough (due to stimulation of auricular branch of vagus) <p><i>Signs</i></p> <ul style="list-style-type: none"> ♦ Brownish-black or yellowish plug of wax ear canal obscuring view of tympanic membrane ♦ Ulceration or granuloma formation (wax granuloma) in long-standing cases ♦ Conductive deafness

Treatment

Conservative

a. Cerumolytic agents

- 2% paradichlorobenzene drops 3 times/day for 3 days

b. Softening of hard wax

- Drops containing 5% sodium bicarbonate with equal part glycerine and water instilled 5–6 times/day for 3–8 days
- Liquid paraffin, olive oil may be tried

Conservative**a. Cerumolytic agents**

- 2% paradichlorobenzene drops 3 times/day for 3 days

b. Softening of hard wax

- Drops containing 5% sodium bicarbonate with equal part glycerine and water instilled 5–6 times/day for 3–8 days
- Liquid paraffin, olive oil may be tried

c. Syringing

- Removing wax from ear using water based on principle of pressure built by liquid in a narrow closed cavity at closed end and return

Procedure

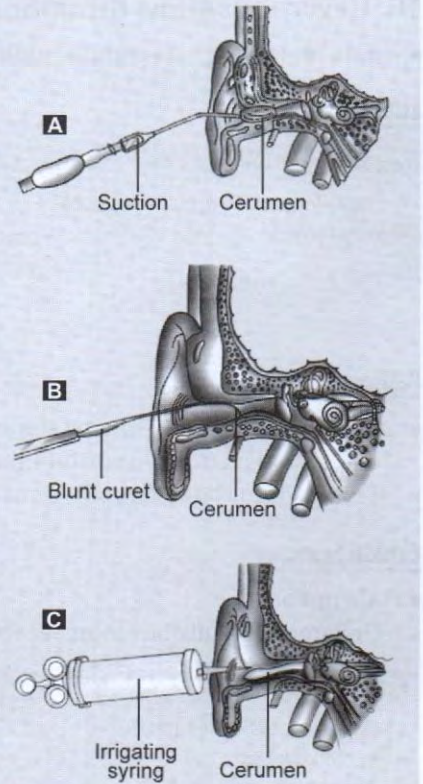
- Make patient to sit or lie down with ear to be syringed towards doctor, slightly tilted downwards to collect return fluid
- Place a towel around his neck
- Ask patient to hold kidney tray over his shoulder or on bed
- Pull pinna upwards and backwards and using aural syringe direct a stream of sterile water (normal saline) at room temperature along posterosuperior wall of meatus
- Wax would be expelled by return gush of water into kidney tray
- Mop dry ear canal and tympanic membrane with cotton and inspect for any residual wax or any trauma
- Instill antibiotic drops into ear canal

d. Instrumental manipulation

- Done by experienced hands under direct supervision

Procedure

- Create a space between wax and meatal wall
- Pass cerumen hook/scoop or Jobson-Horne probe beyond wax
- Drag out whole plug of wax in single piece



Figures 7A to C: Cerumen—treatment modalities

15. Write short note on tympanosclerosis.

- Tympanosclerosis is irreversible degenerative changes in tympanic membrane and middle ear submucosa.

Etiopathogenesis

Etiology	Predisposing factors	Pathology
<ul style="list-style-type: none"> ♦ Imperfect healing of acute or chronic otitis media 	<ul style="list-style-type: none"> ♦ Drying effect of air ♦ Ventilatory tube ♦ Grommet insertion 	<ul style="list-style-type: none"> ♦ Initially hyalinization followed by deposition of plaques of collagen with calcareous deposits in fibrous layer of tympanic membrane and middle ear submucosa

Clinical Features

Symptoms	Signs
<ul style="list-style-type: none"> ♦ History of acute otitis media ♦ Asymptomatic if small ♦ Conductive deafness if involves ossicular chain or oval window 	<ul style="list-style-type: none"> ♦ Chalky white plaques on tympanic membrane (myringosclerosis)

Treatment

Conservative	Operative
<ul style="list-style-type: none"> ♦ No treatment of small plaques ♦ Hearing aids in cases of unsuccessful surgery 	<ul style="list-style-type: none"> ♦ Myringoplasty for myringosclerosis ♦ Excision of tympanosclerotic deposits followed by tympanoplasty including ossiculoplasty if involves ossicles ♦ Stapedectomy or fenestration in case of fixation of foot plate of stapes

16. Cavernous sinus thrombosis.

- Rare and dangerous complication of sinusitis.

Etiology

Causative organisms	Source of infection	Predisposing factors
<ul style="list-style-type: none"> ♦ Staphylococci (coagulase positive) ♦ Streptococci 	<ul style="list-style-type: none"> ♦ Sinusitis, particularly ethmoidal and sphenoidal and less commonly frontal ♦ Infection of dangerous area of face (external nose, vestibule, septum and upper lip) ♦ Orbital cellulitis and abscess 	<ul style="list-style-type: none"> ♦ Immunocompromised patient

Pathogenesis

- Coagulase toxin released by staphylococci enhances coagulation of blood and produces marked changes in intima of vein and changes in surrounding tissue
- This leads to septic thrombosis of cavernous sinus.

Clinical Features

- Abrupt onset
- Unilateral initially but soon becomes bilateral.

Symptoms	Signs
<ul style="list-style-type: none"> ♦ High fever with chills and rigors ♦ Swollen eyelids ♦ Pain around eye ♦ Diminution of vision ♦ Proptosis ♦ Delirium, coma at late stage 	<ul style="list-style-type: none"> ♦ Rapid, small and thready pulse ♦ Chemosis ♦ Total ophthalmoplegia (due to involvement of III, IV and V nerves) ♦ Dilated and fixed pupils ♦ Fixed optic disc with congestion and edema ♦ Raised intraocular pressure ♦ Decreased sensations over distribution of ophthalmic division of V nerve

Investigations	Treatment
a. Blood culture and sensitivity – To identify causative organism and its antibiotic sensitivity b. Fundoscopy – Shows papilledema	<ul style="list-style-type: none"> ♦ Treatment of underlying cause Conservative <ul style="list-style-type: none"> ♦ Antibiotics intravenously to control infection ♦ Anticoagulants ♦ Corticosteroids in severe cases

17. Lingual tonsillitis.

- Lingual tonsils are important component of Waldeyer's lymphatic ring
- Lingual tonsillitis is inflammation of lingual tonsil.

Types

	Acute lingual tonsillitis	Chronic lingual tonsillitis
Etiology	<ul style="list-style-type: none"> ♦ <i>Streptococcus</i> ♦ <i>Staphylococcus</i> ♦ <i>Haemophilus influenzae</i> ♦ <i>Pneumococci</i> 	<ul style="list-style-type: none"> ♦ Compensatory hypertrophy post-tonsillectomy
Clinical features		
Symptoms	<ul style="list-style-type: none"> ♦ Unilateral dysphagia ♦ Feeling of lump in throat 	<ul style="list-style-type: none"> ♦ Lump in throat ♦ Dysphagia ♦ Thick plummy voice

	<i>Acute lingual tonsillitis</i>	<i>Chronic lingual tonsillitis</i>
Signs	<ul style="list-style-type: none"> ♦ Painful movements of tongue ♦ Tender base of tongue ♦ Enlarged and congested lingual tonsils studded with follicles ♦ Enlarged cervical lymph nodes 	
Treatment		
Conservative	♦ Antibiotics (penicillin or erythromycin)	<ul style="list-style-type: none"> ♦ Antibiotics ♦ Avoiding irritant foods
Operative		♦ Diathermy/cryosurgery to reduce size of lingual tonsil
Complications	<ul style="list-style-type: none"> ♦ Edema of epiglottis and larynx ♦ Suppuration 	

18. Cricothyrotomy.

- Cricothyrotomy (laryngotomy) is an emergency procedure of opening airway through cricothyroid membrane.

Procedure

- Extend patients head and neck
- Identify lower border of thyroid cartilage and cricoid ring
- Make vertical incision on skin
- Cut cricothyroid membrane with a transverse incision
- Insert a small tracheostomy tube or insert handle of knife and turn it at right angle to keep this space open if tube is not available.

Postoperative Care

- Perform elective tracheostomy as early as possible.

Complications

- Perichondritis
- Subglottic edema
- Laryngeal stenosis.

19. Subglottic stenosis.

- Subglottic stenosis is narrowing of lumen due to abnormal thickening of cricoid cartilage or fibrous tissue below vocal cords.

<i>Etiology</i>	<i>Pathology</i>
<ul style="list-style-type: none"> ♦ Congenital ♦ Acquired <ul style="list-style-type: none"> – Direct trauma – High tracheostomy – Prolonged intubation 	<ul style="list-style-type: none"> ♦ Abnormal submucosal tissue thickness ♦ Abnormal shaped cricoids cartilage ♦ Cricoids cartilage with small dimension ♦ Posterosuperior displacement of 1st tracheal ring

Clinical Features

- Symptoms become evident in 1st week to 1st month of life (congenital)
- Boys affected twice as females.

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Usually asymptomatic ♦ Recurrent or persistent croup ♦ Dyspnea and stridor (due to upper respiratory tract infections) 	<ul style="list-style-type: none"> ♦ Subglottic diameter <4 mm in full term neonate (normal—4.5–5.5 mm) or <3 mm in premature neonate (normal—3.5 mm)



Figure 8: Subglottic stenosis

Investigations

- X-ray (AP view of neck)
 - Smooth symmetric narrowing of subglotti 2–3 mm below free edge of vocal cords
- Diagnostic laryngoscopy.

Differential Diagnosis

- Laryngeal web
 - White or pink, thick or thin membrane attached anteriorly to both vocal cords with sharp and curved posterior border
- Subglottic hemangioma
 - Unilateral bulge

Treatment		Prognosis
Conservative	Operative	
<ul style="list-style-type: none"> ♦ Treatment of upper respiratory tract infection 	<ul style="list-style-type: none"> ♦ Subglottic dilatation ♦ Tracheostomy for severe stridor ♦ Laryngotracheoplasty 	<ul style="list-style-type: none"> ♦ Improvement in many as larynx grows

20. Compare direct vs indirect laryngoscopy.

- Laryngoscopy is diagnostic technique of visualization of larynx

Comparison of Indirect and Direct Laryngoscopy

Point of comparison	Indirect laryngoscopy	Direct laryngoscopy
♦ Visualization	Inverted mirror image	Direct visualization
♦ Appearance of cords	Vocal cords and false cords appear to be in one plane (flattened appearance)	Vocal cords and false cords seen in true perspective
♦ Vocal cord movements	Better appreciated	Seen only if performed under anesthesia (local or light general)
♦ Setup	OPD procedure	OT procedure
♦ Visualization of anterior commissure, ventricle, subglottic	Not satisfactory	Satisfactory
♦ Foreshortening of vertical axis	Present	Absent
♦ Application	Good for diagnosis, may be useful for biopsy and removal of foreign bodies	Useful for diagnosis, biopsy and surgical procedures

21. Describe the types of mastoidectomies.

- Mastoidectomy is removal of mastoid air cells done for any chronic infection of mastoid air cells or any complication of chronic otitis media involving mastoid.

Classification (Types)

- Simple mastoidectomy (Cortical or conservative mastoidectomy/ Schwartz operation)
 - Indicated for acute coalescent mastoiditis
 - Aims to drain mastoid only
 - Consists of exenteration of all accessible mastoid air cells preserving posterior meatal wall
 - Does not affect hearing
- Radical mastoidectomy
 - Indicated for chronic mastoiditis with cholesteatoma affecting attic and mesotympanum
 - Consists of exenteration of disease from middle ear, attic and mastoid with removal of diseased ossicles (except stapes footplate), tympanic membrane and mucoperiosteal lining thus converting mastoid, attic and middle ear and external ear into single cavity
 - Results in conductive deafness
- Modified radical mastoidectomy
 - Indicated for chronic mastoiditis with cholesteatoma affecting attic but sparing mesotympanum
 - Consists of extenteration of disease from attic and mastoid by removal of posterior meatal and lateral wall and converting mastoid, attic and external ear into single cavity
 - Tympanic membrane remnant, functioning ossicles and reversible mucosa and function of Eustachian tube are preserved
 - Does not affect hearing

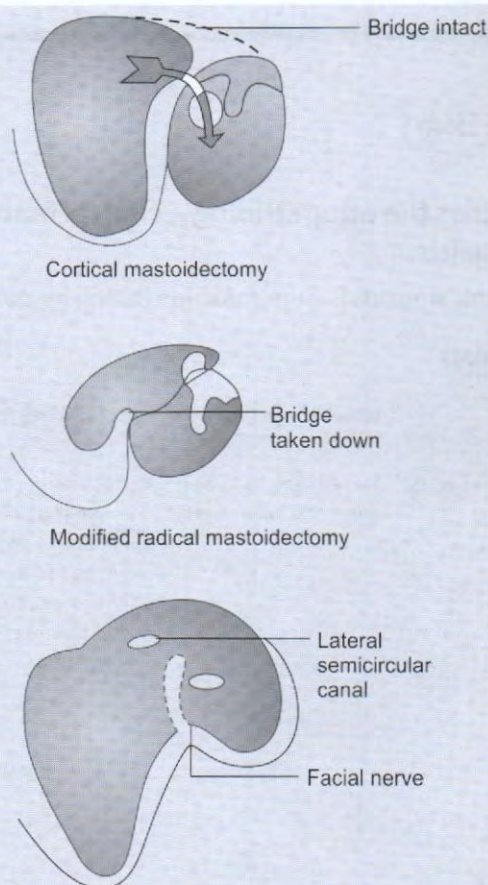


Figure 9: Mastoidectomies—types

22. Enumerate complications of radiotherapy.

- Radiotherapy is an important modality of treating head and neck cancers.

Complications

Early (MOM'S HAIRS)	Late (DR'S PROTECT)
<ul style="list-style-type: none"> Mucositis Edema of larynx Mucosal dryness Skin reactions (erythema, dry or wet desquamation) Hemoatopoietic suppression Acute transverse myelitis Infection by Candida Radiation sickness (nausea, vomiting, loss of appetite) Sialadenitis 	<ul style="list-style-type: none"> Decaying of teeth Radiation retinopathy and cataract Skin changes (atrophy, subcutaneous fibrosis) Permanent xerostomia Radiation induced malignancy (thyroid carcinoma, osteosarcoma of orbit) Osteoradionecrosis Trismus (due to fibrosis of TM joint and muscles) Endocrinal deficiency (pituitary, thyroid) Chronic sialadenitis Transverse myelitis

SAMPLE PAPERS

MBBS PHASE III EXAMINATION

SAMPLE PAPER-2

LONG ESSAYS

1. Discuss the etiopathology, clinical features and management of chronic frontal and maxillary sinusitis.

- Chronic sinusitis is sinus infection lasting for months or years.

Etiopathology

Cause	Causative organisms	Pathophysiology (vicious cycle)	Pathology (simultaneous processes of destruction and healing)
Unresolved acute sinusitis	Mixed aerobic and anaerobic organisms	<ul style="list-style-type: none">• Destruction of normal ciliated epithelium impairing sinus drainage• Pooling and stagnation of secretions in sinus which predisposed to infection• Persistence of infection causes mucosal changes like loss of cilia, edema and polyp formation	<ul style="list-style-type: none">• Sinus mucosa thickens and becomes polypoidal (hypertrophic sinusitis) or undergoes atrophy (atrophic sinusitis)• Desquamation, regeneration or metaplasia of surface epithelium• Infiltration of submucosa with lymphocytes and plasma cells along with presence of microabscesses, granulations, fibrosis or polyp formation

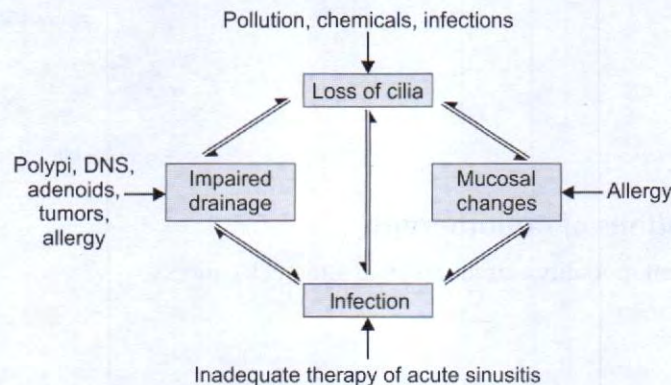


Figure 1: Pathophysiology of chronic sinusitis

Clinical Features (Similar but Less Severe to Acute Sinusitis)

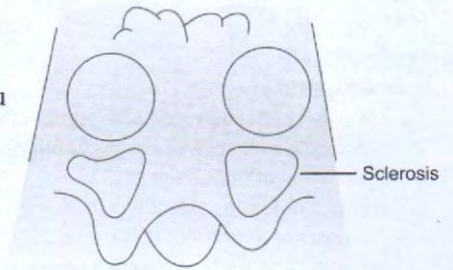
- Purulent nasal discharge (most common)—foul-smelling in anaerobic infection
- Local pain and headache in acute exacerbations
- Nasal stuffiness and anosmia.

Investigations

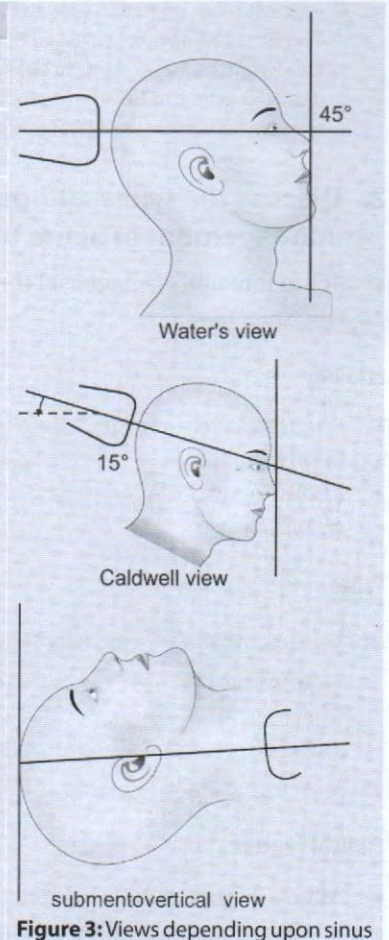
- Demonstration of pus in sinus by aspiration and irrigation (confirmatory)
- Culture and sensitivity of sinus discharge.

Radiological Evaluation (Important Tool)**a. X-ray**

- Plain X-ray of involved sinus shows mucosal thickening or opacity and flu
- Contrast X-ray shows soft tissue changes in sinus mucosa.

**Figure 2: Chronic sinusitis X-ray PNS****Views (depending upon sinus)**

Views	Position	Ideal to see
♦ Occipitomental view (Water's view)	Nose and chin with open mouth in contact with film	Maxillary sinus and frontal sinus
♦ Occipitofrontal view (Caldwell view)	Forehead and tip of nose in contact with film	Frontal sinus and maxillary antrum
♦ Submentovertical view	Fully extended neck with vertex in contact with film	Sphenoidal sinus, ethmoidal sinuses and posterior wall of maxillary sinus
♦ Lateral oblique view	Lateral side of head in contact with film	Ethmoidal sinuses

**Figure 3: Views depending upon sinus****b. CT scan**

- Investigation of choice
- Provides excellent evaluation particularly of sphenoidal and ethmoidal sinuses.

Treatment**Frontal sinusitis****Conservative**

- ♦ Antibiotics, decongestants, antihistaminics and sinus irrigations

Operative treatment

- ♦ Treatment of choice in such cases as drains pus and exudates and helps removing diseased mucosa thus favoring recovery of normal sinus mucosa

Maxillary sinusitis

Frontal sinusitis**Operative options**

- a. Intranasal drainage
 - By either correction of deviated septum, removal of a polyp or anterior portion of middle turbinate, or intranasal ethmoidectomy
- b. Trephination of frontal sinus
- c. External frontoethmoidectomy (Howarth's or Lynch operation)
 - Consists of removal of diseased mucosa, exenteration of ethmoid cells and creating a new frontonasal duct by entering frontal sinus through its floor by a curvilinear incision round the inner margin of the orbit
- d. Osteoplastic flap operation (unilateral or bilateral)
 - Consists of removal of diseased tissues and drainage of sinus through newly created frontonasal duct or obliteration of sinus with fat by stripping off diseased as well as healthy mucosa through a coronal or a brow incision and reflecting anterior wall of frontal sinus as an inferior osteoplastic flap

Maxillary sinusitis**Operative options**

- a. Antral puncture and irrigation
 - Consists of removal of pus and exudates followed by irrigation of sinus cavity using a cannula inserted through inferior meatus
- b. Intranasal antrostomy
 - Consists of provision for aeration and free drainage of sinus cavity by creating a window in inferior meatus near floor of antrum
- c. Caldwell-Luc operation (radical operation)
 - Consists of drainage of pus and exudates through anterior approach (sublabial incision) followed by creating a window between antrum and inferior meatus

2. Discuss the types, etiopathology, clinical diagnosis and management of acute tonsillitis.

- Acute tonsillitis is bacterial infection of tonsils either primarily or secondary to viral infection.

Etiology

- *Streptococcus*—Group A beta hemolytic (most common)
- Staphylococci
- Pneumococci
- *H. influenzae*

Types

a. Acute catarrhal or superficial tonsillitis	Seen as a component of generalized pharyngitis and mostly seen in viral infections
b. Acute follicular tonsillitis	Spread of infection into crypts which become filled with purulent material, presenting at their openings as yellowish spots
c. Acute parenchymatous tonsillitis	Involvement of tonsillar substance which are uniformly enlarged and red
d. Acute membranous tonsillitis	Coalescence of cryptic exudation to form a membrane on the surface of tonsil

Clinical Features

- Affects school-going children (peak 5–6 years) often but also seen in adults
- Rare in infants and elderly (above 50 years of age).

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Throat pain, dry throat ♦ Difficulty in swallowing due to local pain ♦ Fever (varying from 38°C to 40°C) and may be associated with chills and rigors ♦ Earache—either referred pain or due to complicating acute otitis media ♦ Constitutional symptoms include headache, general body aches, malaise and constipation ♦ Abdominal pain due mesenteric lymphadenitis 	<ul style="list-style-type: none"> ♦ Dry coated tongue and foetid breath (halitosis) ♦ Hyperemia of pillars, soft palate and uvula ♦ Red and swollen tonsils ♦ Purulent material may present as yellowish spots at opening of crypts (acute follicular tonsillitis) ♦ Whitish membrane may be present on medial surface of tonsil which can be easily wiped away with a swab (acute membranous tonsillitis) ♦ Congested and enlarged tonsils, which may meet in midline along with edema of uvula and soft palate (acute parenchymatous tonsillitis) ♦ Enlarged and tender jugulodigastric lymph nodes



Figure 4: Acute sinusitis

Investigations

- a. Rapid strep tests
 - Latex agglutination or ELISA methods extract antigen (group-A streptococcal) from a swab—highly specific (95%), but less sensitive (60–100%).
- b. Throat culture
 - Culture from swab from posterior pharynx and tonsillar area.

Treatment

Conservative

- Bed rest with plenty of fluids
- Analgesics to relieve local pain and bring fever down
- Antibiotics for 7–10 days
 - Penicillin or amoxicillin (drug of choice)
 - Erythromycin in patients allergic to penicillin.

Complications

- Chronic tonsillitis with recurrent acute attacks
 - Due to incomplete resolution
 - Chronic infection may persist in lymphoid follicles of tonsil in form of microabscesses
- Peritonsillar abscess
- Parapharyngeal abscess
- Cervical abscess due to suppuration of jugulodigastric lymph nodes
- Acute otitis media—seen in recurrent acute tonsillitis
- Rheumatic fever—associated with Group A beta-hemolytic streptococci
- Acute glomerulonephritis (rare)
- Subacute bacterial endocarditis—usually due to *Streptococcus viridans* infection.

SHORT ESSAYS

3. Bilateral nasal obstruction—differential diagnosis.

- Bilateral nasal obstruction is an important differential diagnosis of ethmoidal polyps.

Differential Diagnosis

Vestibule	Nasal cavity	Nasopharynx
<ul style="list-style-type: none"> ♦ Bilateral vestibulitis ♦ Collapsing nasal alae ♦ Stenosis of nares ♦ Congenital atresia of nares 	<ul style="list-style-type: none"> ♦ Acute rhinitis (viral/bacterial) ♦ Chronic rhinitis and sinusitis ♦ Rhinitis medicamentosa ♦ Allergic rhinitis ♦ Hypertrophic turbinates ♦ Deviated nasal turbinates ♦ Nasal polypi ♦ Atrophic rhinitis ♦ Rhinitis sicca ♦ Septal hematoma ♦ Septal abscess ♦ Bilateral choanal atresia 	<ul style="list-style-type: none"> ♦ Adenoid hyperplasia ♦ Large choanal polyp ♦ Thornwaldt's cyst ♦ Adhesion between soft palate and posterior pharyngeal wall ♦ Large benign and malignant tumors

4. Vincent's angina.

- An acute ulcerative lesion of pharynx involving one or both tonsils
- Also called trench mouth, acute membranous pharyngitis or tonsillitis.

Etiology

<i>Causative organisms</i>	<i>Predisposing factors</i>
<ul style="list-style-type: none"> ♦ Fusiform bacillus (Vincent's organism) ♦ <i>Spirochete denticola</i> 	<ul style="list-style-type: none"> ♦ Poor dental hygiene ♦ Caries teeth ♦ Pyorrhea ♦ Overcrowding ♦ Poor diet ♦ General debility

Clinical Features

- Usually affects young adults and middle aged people
- Sudden onset.

<i>Symptoms</i>	<i>Signs</i>
<ul style="list-style-type: none"> ♦ High grade fever ♦ Severe pain in throat ♦ Bad smell from mouth (fishy odor) 	<ul style="list-style-type: none"> ♦ Affected area covered with greyish membranous slough which on removal causes considerable loss of tissue leaving behind bleeding base ♦ Slough reforms after removal ♦ Cervical lymphadenitis

Investigations

- Pharyngeal swab
 - Gram staining and culture
 - Hanging drop preparation.

Treatment

<i>Conservative</i>	<i>Operative</i>
<ul style="list-style-type: none"> ♦ Systemic broad-spectrum antibiotics (metronidazole and clindamycin) ♦ Analgesics to relieve pain and fever ♦ Antiseptic mouth wash ♦ Antiseptic paint over fauces and gum 	<ul style="list-style-type: none"> ♦ Surgical debridement

5. How would you diagnose and treat tonsillar diphtheria?

- Tonsillar diphtheria is involvement of tonsils in faucial diphtheria.

Etiology

<i>Causative organism</i>	<i>Source of infection</i>	<i>Spread of infection</i>	<i>Incubation period</i>
• <i>Corynebacterium diphtheriae</i>	• Carriers	• Droplets	• 2–6 days (slow in onset)

Clinical Features

- More common in children

<i>Symptoms</i>	<i>Signs</i>
<ul style="list-style-type: none"> ♦ Fever (does not rise above 38°C) ♦ Toxic look 	<ul style="list-style-type: none"> ♦ Membrane over tonsil <ul style="list-style-type: none"> – Grayish white membrane – Tenacious and adherent to tonsil – On removal causes bleeding leaving behind raw areas – Spreading over soft palate and posterior pharyngeal wall – Enlarged and tender cervical lymph nodes especially jugulodigastric ♦ Bull neck appearance (due to enlarged cervical lymph nodes) ♦ Tachycardia with weak pulse

Investigations

- Staining with Albert's stain (rapid diagnostic test)
- Culture and sensitivity for 48 hours
- Urine shows albuminuria.

Treatment

- Started at suspicion of diphtheria.

Objective

- Neutralize circulating free exotoxin in blood
- Kill exotoxin producing microorganisms.

Supportive

- Bed rest
- Plenty of fluid.

Specific**a. Antitoxin**

Dose		Route	Precautions
20,000–40,000 units	80,000–120,000 units	♦ IV infusion in saline in 60 minutes	♦ Sensitivity testing to horse serum by conjunctival or intracutaneous test by diluted antitoxin
♦ For diphtheria <48 hours	♦ For diphtheria >48 hours		
♦ For membrane confined to tonsils	♦ For extensive membrane		

b. Antibiotics

Benzyl penicillin	Erythromycin
♦ 600 mg QID IM for 1 week	♦ 500 mg QID orally for 1 week

Complication

Respiratory	Cardiac	Neurological (appear after few weeks)
♦ Airway obstruction by membrane	♦ Myocarditis, cardiac arrhythmias and acute circulatory failure	♦ Paralysis of soft palate, diaphragm and ocular muscles

6. Tuberculosis of larynx.

- Tuberculosis of larynx is usually secondary to pulmonary tuberculosis.

Etiopathogenesis

Causative organism	Source of infection	Spread of infection	Pathology	Affected parts (in descending order)
<i>Mycobacterium tuberculosis</i>	Pulmonary tuberculosis	♦ Bronchogenic spread: Tubercle bacilli, carried by sputum from bronchi, settle and penetrate intact laryngeal mucosa particularly in interarytenoid region ♦ Hematogenous spread ♦ Lymphatic spread	♦ In the larynx, bacilli leads to formation of submucosal tubercles which later caseate and ulcerate ♦ Laryngeal mucosa appears red and swollen due to cellular infiltration (pseudodema)	♦ Interarytenoid fold ♦ Ventricular bands ♦ Vocal cords ♦ Epiglottis Posterior part is affected more than anterior

Clinical Features

- Commonly affects middle aged males.

Symptoms	Signs
<ul style="list-style-type: none"> History of pulmonary tuberculosis symptoms (cough, chest pain, evening rise of fever, etc.) Weakness of voice (earliest) Hoarseness Severe pain radiating to ear (due to ulceration in larynx) Painful swallowing initially to liquids (due to exposed nerve endings in ulcers) Marked dysphagia later Hemoptysis in advanced cases 	<ul style="list-style-type: none"> Impairment of adduction—early sign of tuberculosis Hyperemia of vocal cord (whole extent or posterior part) Swelling in interarytenoid region with mamillated appearance Ulceration of vocal cord with mouse nibbled appearance Superficial ragged ulceration on arytenoids and interarytenoid region Granulation tissue in interarytenoid region or vocal process of arytenoids Pseudoedema of epiglottis (turban epiglottis) Swelling of ventricular bands and aryepiglottic folds Marked pallor of surrounding mucosa

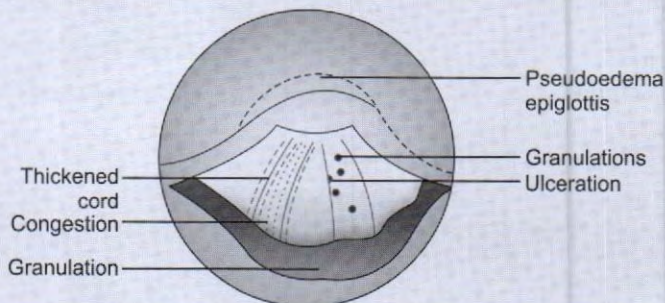


Figure 5: Tuberculous larynx

Investigations

- X-ray chest, Mantoux test and sputum examination: To detect pulmonary tuberculosis
- Biopsy of laryngeal lesion: To exclude carcinoma and differential diagnosis (syphilis, nonspecific chronic laryngitis).

Treatment

- Voice rest
- Antitubercular treatment as per RNTCP.

7. Speech rehabilitation following laryngectomy.

- Complete loss of speech of after laryngectomy requires speech rehabilitation of patient to place him in functional society.

Techniques

- Written language
 - Using pen and paper, chalk and slate, etc.
- Aphonic lip speech (with sign language)
 - By trapping air in buccal cavity
- Esophageal speech (voice)
 - Speech produced by slowly ejecting (burping) swallowed air into pharynx from upper esophagus where it is held
 - Instituted 3rd-6th week postoperatively.

Advantages	Disadvantages
<ul style="list-style-type: none"> Simple and inexpensive Loud and understandable speech 	<ul style="list-style-type: none"> Requires lot of training and practice Produces only 6–10 words at one go Rough voice

d. Artificial larynx

Indications

- Failure to learn esophageal speech

Techniques

i. Electrolarynx

- A small transistorized, battery operated portable device used to produce speech by holding its vibrating disk against soft tissues of neck which produces low pitched sound in hypopharynx which is further modified into speech by tongue, teeth, palate and lips

ii. Transoral pneumatic device

- Produces sound by using expired air to vibrate a rubber diaphragm, vibrations of which are carried to back of oral cavity by a rubber tube to be converted to sound using modulators.

Advantages	Disadvantages
♦ Does not require any training or second surgery	♦ Expensive ♦ Metallic voice

e. Tracheoesophageal speech (Neoglottis formation)

- Produces sound by carrying air from trachea to esophagus or hypopharynx through a skin lined fistula or artificial prosthesis (Blom-Singer or Panje) which is converted to speech by modulators.

Advantages	Disadvantages
♦ Better controlled speech ♦ Can speak more words at a time	♦ Food entering into trachea ♦ Stenosis of fistula

8. Achalasia cardia.

- Achalasia cardia is condition characterized by failure of relaxation of lower esophageal sphincter for passage of food
- Also called **cardiospasm**

Etiology (theories of causation)	Pathology
<ul style="list-style-type: none"> ♦ Degeneration of Auerbach's plexus causing failure of relaxation of cardiac orifice (Hurst) ♦ Degeneration in utero due to ischemia <u>during rotation of gut</u> ♦ Abnormal pinch-cock action of right crus of diaphragm (Jackson) ♦ Chaga's disease (trypanosomiasis) 	<ul style="list-style-type: none"> ♦ Spasm of lower esophageal sphincter due to neuromuscular incoordination

Clinical Features

- Usually affects at **young** age
- **Insidious** onset.

Symptoms	Signs
<ul style="list-style-type: none"> ♦ Retrosternal or epigastric fullness following meals ♦ Difficulty to swallowing (dysphagia) more to liquids than solids ♦ Regurgitation of swallowed food particularly at night ♦ Loss of weight 	<ul style="list-style-type: none"> ♦ Pooling of saliva on indirect laryngoscopy

Investigations

a. **Radiography (barium swallow)**

- Fusiform dilatation of esophagus with narrowed rat tail lower end (bird beak appearance)
- Fluid levels may be seen
- Absence of gas shadow in fundus

b. **Manometric studies**

- Demonstrates low pressure in body of esophagus and high pressure at lower sphincter and failure of sphincter to relax

c. **Endoscopy**

- To exclude benign strictures or malignant change.

Treatment

<i>Conservative</i>	<i>Operative</i>
a. Smooth muscle relaxants <ul style="list-style-type: none"> – Isosorbide dinitrate, amyl nitrate, calcium channel blockers before meals (tried earlier) 	a. Modified Heller's operation or cardiomyotomy (treatment of choice) <ul style="list-style-type: none"> – Myotomy of narrowed lower portion of esophagus to relieve spasm
b. Injection of botulinum toxin type A <ul style="list-style-type: none"> – To relax lower end of esophagus 	b. Forceful pneumatic dilatation of lower esophagus using Hurst mercury bougies <ul style="list-style-type: none"> – In patients unfit for surgery
	c. Anastomosis between stomach and esophagus <ul style="list-style-type: none"> – If esophagus grossly lengthened or kinked

Complications

- **Benign strictures**
- **Malignant** transformation.

9. Clinical features and management of foreign bodies in esophagus.

- Impaction of foreign bodies like fish bones, pins, coins, dentures, etc. is very common.

Etiology

<i>Predisposing factors</i>	<i>Site of lodgment</i>
<ul style="list-style-type: none"> ♦ Use of dentures: <ul style="list-style-type: none"> – Prevents tactile sensation ♦ Loss of protective mechanism <ul style="list-style-type: none"> – Unconsciousness, seizures, deep sleep, intoxication ♦ Carelessness <ul style="list-style-type: none"> – Poorly cooked food, improper mastication, hasty eating ♦ Narrowed esophageal lumen <ul style="list-style-type: none"> – Due to stricture or carcinoma ♦ Psychosis <ul style="list-style-type: none"> – Ingestion of foreign body to commit suicide 	<ul style="list-style-type: none"> ♦ At or just below cricopharyngeal sphincter (commonest site) ♦ Broncho-aortic constriction ♦ Cardiac end of esophagus ♦ Upper esophagus just below cricopharyngeal sphincter

Clinical Features

- Usually common at extreme of ages.

<i>Symptoms</i>	<i>Signs</i>
<ul style="list-style-type: none"> ♦ History of initial choking or gagging ♦ Discomfort or pain just above clavicle on right or left of trachea which increases on attempt to swallow ♦ Difficulty in swallowing (dysphagia) due to partial or total obstruction ♦ Drooling of saliva ♦ Respiratory distress (due to compression of trachea and laryngeal edema) ♦ Substernal or epigastric pain (due to esophageal spasm or incipient perforation) 	<ul style="list-style-type: none"> ♦ Tenderness in lower part of neck on right or left of trachea ♦ Pooling of saliva in pyriform fossa ♦ Protruding foreign body in postcricoid region

Investigations

- X-ray (Lateral view of neck, PA and lateral view of chest)
 - Show presence and location of radiopaque foreign body
 - Prevertebral widening
 - Anteriorly displaced airway

- b. Fluoroscopy
 - Observation of barium containing capsule or cotton passing through esophagus to detect radiolucent foreign bodies
- c. Esophagoscopy
 - To confirm and remove foreign body.

Treatment

Conservative	Operative (removal of foreign body)
<ul style="list-style-type: none"> ♦ Antibiotics intravenously ♦ Analgesics ♦ Parenteral nutrition ♦ Steroids 	<p>Techniques</p> <ul style="list-style-type: none"> ♦ Through rigid esophagoscopy under local or general anesthesia ♦ Through esophagotomy by external route <p>Indications</p> <ul style="list-style-type: none"> ♦ Pain and tenderness at site of lodgment ♦ Nonprogressive foreign body ♦ Foreign body >5 cm in children under 2 years ♦ Pyloric stenosis <p>Approach</p> <ul style="list-style-type: none"> ♦ Cervical ♦ Transthoracic

Complications

- Respiratory obstruction (due to tracheal compression or laryngeal edema)
- Esophagitis
- Perioesophageal cellulitis and abscess in neck
- Perforation of esophagus, aorta
- Tracheoesophageal fistula
- Ulceration and stricture.

10. Indications and contraindications of rigid laryngoscopy.

- Rigid laryngoscopy or direct laryngoscopy is direct visualization of larynx and hypopharynx

Indications		Contraindications
Diagnostic	Therapeutic	
<ul style="list-style-type: none"> ♦ Diagnosis and evaluation of laryngeal diseases if indirect laryngoscopy is not possible (as in infants, children) or is unsuccessful (due to excessive gag reflex, overhanging epiglottis, etc) ♦ Further evaluation of laryngeal pathology seen on indirect laryngoscopy ♦ Examination of obscured areas ♦ Vocal cord paralysis ♦ To assess extent of growth and take biopsy ♦ To assess laryngeal trauma ♦ As part of bronchoscopy or esophagoscopy ♦ As part of panendoscopy for unknown primary with cervical metastasis 	<ul style="list-style-type: none"> ♦ Removal of benign lesions of larynx (cordal nodules, vocal polyps, cysts) ♦ Foreign body removal from larynx and hypopharynx ♦ Removal of thickened secretions and crust ♦ Injection of telfon paste in vocal cord for vocal cord palsy ♦ Dilatation of laryngeal strictures ♦ For endotracheal intubation 	<ul style="list-style-type: none"> ♦ Cervical spine pathology ♦ Trismus ♦ Long incisor teeth ♦ Laryngeal spasms and stridor ♦ Moderate or marked dyspnea ♦ Short and thick neck ♦ Vascular tumors like aneurysms of aorta ♦ Recent coronary occlusion or cardiac decompensation ♦ General contraindications (anemia, diabetes, hypertension)

Anesthesia

- General anesthesia with endotracheal intubation.

Position

- Supine position with elevated head (10–15 cm) by placing pillow under occiput thus flexing neck at thorax and extending head at atlanto-occipital joint (Boyce's position or Barking dog position).

Procedure

- Place a piece of gauze on upper teeth to prevent injury to them
- Lubricate laryngoscope with autoclaved liquid paraffin or jelly
- Use left hand to hold handle of laryngoscope while right hand retracts lips and guides it into larynx and also handles suction and instruments
- Introduce laryngoscope into oral cavity through right side of angle of mouth pushing tongue to opposite side till base of tongue is reached
- Lift laryngoscope forwards and move it in midline to bring epiglottis in view
- Guide laryngoscope behind epiglottis and lift laryngoscope forward (do not lever it on upper teeth or jaw)—engagement of epiglottis
- Advance tip of laryngoscope for 1cm between ventricular bands (false vocal cords) to examine ventricles and anterior commissure
- Observe mobility of vocal cords
- Then pass is between vocal cords to examine subglottic region
- Undersurface of vocal cords and walls of subglottis can be visualized using right angled telescope
- Withdraw laryngoscope in reverse fashion.

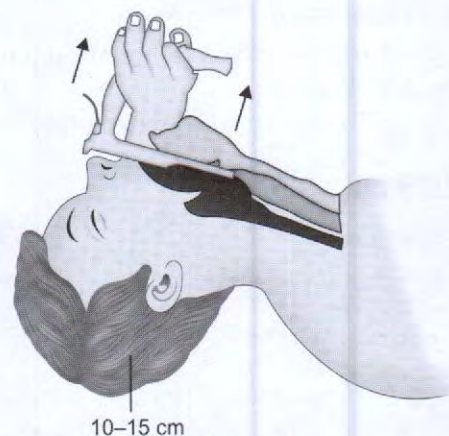


Figure 6: Position of bronchoscopy

Structures Seen (Above Downwards)

Oropharynx	Larynx	Laryngopharynx
<ul style="list-style-type: none"> ♦ Posterior 1/3rd of tongue ♦ Valleculae 	<ul style="list-style-type: none"> ♦ Epiglottis ♦ Aryepiglottic fold ♦ Arytenoid cartilage ♦ False vocal cords ♦ Anterior and posterior commissure ♦ Ventricles ♦ True vocal cords ♦ Subglottis 	<ul style="list-style-type: none"> ♦ Posterior pharyngeal wall ♦ Pyriform fossa ♦ Postcricoid region

Postoperative Care

- Keep patient in coma position to prevent aspiration of blood or secretions
- Watch patient's respiration for laryngeal spasm and cyanosis.

Complications	Advantages	Disadvantages
<ul style="list-style-type: none"> ♦ Injury to lips, tongue, teeth and vocal cords ♦ Trauma to larynx on repeated attempts ♦ Bleeding from operative site ♦ Laryngeal edema particularly of subglottis ♦ Hypoxia and cardiac arrest ♦ Dislocation of cervical vertebrae (if diseased) ♦ Slipping of foreign body or disintegration of foreign body 	<ul style="list-style-type: none"> ♦ Direct visualization ♦ False cord and true cord seen in true perspective ♦ Complete view of larynx ♦ No foreshortening of vertical axis ♦ Possible in infants and children 	<ul style="list-style-type: none"> ♦ Invasive procedure ♦ Requires anesthesia ♦ Needs to be done in operation theater

11. What are the causes of unilateral nasal discharge?

- Nasal discharge is usually conveyed backwards to nasopharynx by ciliary action and swallowed
- However, in certain conditions, nasal discharge may appear at anterior nares.

Causes (Differential Diagnosis) of Unilateral Nasal Discharge (FRANC UU)

a. Foreign body	<ul style="list-style-type: none"> ♦ Foul smelling blood stained discharge ♦ Common in children ♦ History of insertion of foreign body may be present ♦ Foreign body seen on anterior rhinoscopy ♦ Only radiopaque foreign bodies detected on radiography
b. Rhinolith	<ul style="list-style-type: none"> ♦ Foul smelling blood stained discharge ♦ Common in adults ♦ Hard, grayish brown or greenish black, irregular mass seen on anterior rhinoscopy ♦ Detected on radiography
c. Unilateral choanal atresia	<ul style="list-style-type: none"> ♦ Excessive nasal discharge ♦ Complete blockage of nostril ♦ Usually congenital ♦ Diagnosed by probing or radiography
d. Unilateral sinusitis	<ul style="list-style-type: none"> ♦ Mucoïd discharge initially but soon becomes purulent ♦ Nasal obstruction on affected side due to congestion and edema ♦ Constitutional symptoms like malaise, fever and headache ♦ Edema of affected area and tenderness over affected sinus
e. Antrochoanal polyp	<ul style="list-style-type: none"> ♦ Clear discharge (if allergic) or purulent discharge (if infected) ♦ Common in children and young adults ♦ Present as unilateral blocking of nose ♦ Single polypoid dumbbell shaped, large, smooth grayish mass evident on posterior rhinoscopy
f. CSF rhinorrhea	<ul style="list-style-type: none"> ♦ Clear watery discharge containing glucose ♦ Discharge does not contain mucus (handkerchief soaked in it does not stiffen on drying) ♦ Intermittent or continuous discharge ♦ Uncontrollable and cannot be sniffed back ♦ Increase in discharge on sneezing, coughing or bending head forward ♦ Common after head injuries ♦ Confirmed by Dye test and CT scan
g. Neoplasm	<ul style="list-style-type: none"> ♦ Blood stained discharge ♦ Common in elderly

12. Management of acute maxillary sinusitis.

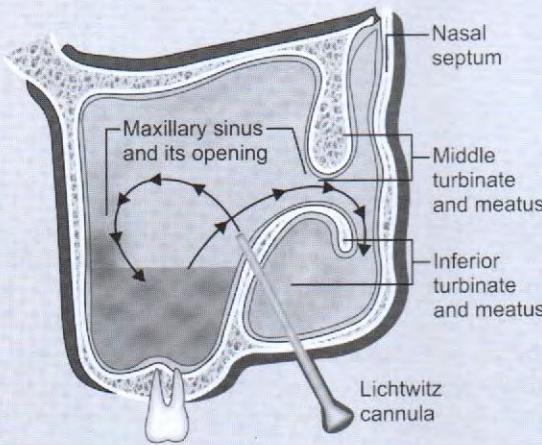
- Acute sinusitis is acute inflammation of sinus mucosa
- Commonly involved sinuses are maxillary, ethmoid, frontal and sphenoid (in descending order).

Types

- Open: Discharge can drain freely into nasal cavity through natural ostia
- Closed: Discharge locked inside the sinuses; causes more severe symptoms and more likely to cause complications.

	Maxillary sinusitis	Frontal sinusitis
Etiology		
Causative organism	<ul style="list-style-type: none"> ♦ Most cases as viral infections followed by bacterial invasion Bacteria involved <ul style="list-style-type: none"> ♦ <i>Streptococcus pneumoniae</i> ♦ <i>Haemophilus influenzae</i> ♦ <i>Moraxella catarrhalis</i> ♦ <i>Streptococcus pyogenes</i> ♦ <i>Staphylococcus aureus</i> ♦ <i>Klebsiella pneumoniae</i> 	

	Maxillary sinusitis	Frontal sinusitis
Etiology		
Exciting causes	<ul style="list-style-type: none">♦ Nasal infections<ul style="list-style-type: none">– Viral rhinitis (most common) and bacterial invasion– Nasal infections travel directly into sinus by continuity or by way of submucosal lymphatics♦ Swimming and diving<ul style="list-style-type: none">– Lead to entry of infected water through ostia– High chlorine content in swimming pools—chemical inflammation♦ Trauma<ul style="list-style-type: none">– Compound fractures or penetrating injuries permit direct infection♦ Barotrauma♦ Dental infections (maxillary sinusitis)<ul style="list-style-type: none">– Infection from molar or premolar teeth or their extraction	
Predisposing causes		
Local	<ul style="list-style-type: none">♦ Obstruction to sinus ventilation and drainage<ul style="list-style-type: none">– Nasal packing– Deviated septum– Hypertrophic turbinates– Edema of sinus ostia due to allergy or vasomotor rhinitis– Nasal polypi– Structural abnormality of ethmoidal air cells– Benign or malignant neoplasm♦ Stasis of secretions<ul style="list-style-type: none">– Secretions of nose may not drain into the nasopharynx because of their viscosity (cystic fibrosis) or obstruction (enlarged adenoids, choanal atresia) and get infected♦ Previous attacks of sinusitis	
General	<ul style="list-style-type: none">♦ Environment<ul style="list-style-type: none">– Common in cold and wet climate– Atmospheric pollution, smoke, dust and overcrowding also predisposes♦ Poor general health<ul style="list-style-type: none">– Recent attack of exanthematous fever (measles, chickenpox, whooping cough), nutritional deficiencies and systemic disorders (diabetes, immune deficiency syndromes)	
Pathology	<ul style="list-style-type: none">♦ Hyperemia, exudation of fluid, outpouring of polymorphonuclear cells and increased activity of serous and mucous glands♦ Disease may be mild (nonsuppurative) or severe (suppurative)—depending upon virulence of organism, defences of host and natural sinus drainage♦ Exudate is serous which becomes mucopurulent or purulent♦ Mucosal lining may be destroyed in severe infection♦ Failure of ostium to drain results in empyema of sinus and destruction of its bony walls leading to complications	
Clinical features		
Symptoms	<ul style="list-style-type: none">♦ Constitutional symptoms like fever, general malaise and body ache (due to toxemia)♦ Headache confined to forehead♦ Pain over upper jaw but may be referred to gums or teeth and is aggravated by stooping, coughing or chewing	<ul style="list-style-type: none">♦ Frontal headache<ul style="list-style-type: none">– Usually severe and localized over affected sinus– Shows characteristic periodicity, i.e. comes up on waking, gradually increases and reaches its peak by about mid day and then starts subsiding (Office headache—present only during office hours)
Signs	<ul style="list-style-type: none">♦ Tenderness over anterior wall of antrum on pressure or tapping♦ Redness and edema of cheek♦ Lower eyelid may become puffy♦ Anterior rhinoscopy/nasal endoscopy<ul style="list-style-type: none">– Reveals pus or mucopus in middle meatus– Mucosa of middle meatus and turbinate appear red and swollen	<ul style="list-style-type: none">♦ Tenderness on upward pressure on floor of frontal sinus, just above medial canthus or tapping over anterior wall in medial part of supraorbital region♦ Edema of upper eyelid with suffused conjunctiva and photophobia♦ Anterior rhinoscopy/nasal endoscopy<ul style="list-style-type: none">– A vertical streak of mucopus is seen high up in anterior part of middle meatus

	Maxillary sinusitis	Frontal sinusitis
Etiology		
Investigations	<ul style="list-style-type: none"> Posterior rhinoscopy/nasal endoscopy <ul style="list-style-type: none"> Pus seen on upper soft palate (postnasal discharge) a. Postural test <ul style="list-style-type: none"> In absence of pus in middle meatus, it is decongested and patient is made to sit with affected sinus turned up. Examination after 10–15 min may show discharge in middle meatus b. Transillumination test <ul style="list-style-type: none"> Opacity of affected sinus c. X-rays (Waters' view) <ul style="list-style-type: none"> Opacity or a fluid level in involved sinus d. CT scan <ul style="list-style-type: none"> Investigation of choice 	<ul style="list-style-type: none"> Inflamed nasal mucosa in middle meatus a. X-rays (Waters' view and lateral view) <ul style="list-style-type: none"> Opacity or a fluid level in involved sinus b. CT scan <ul style="list-style-type: none"> Investigation of choice
Treatment		
Conservative	<ul style="list-style-type: none"> a. Antibiotics <ul style="list-style-type: none"> Ampicillin and amoxicillin Amoxicillin + clavulanic acid or cefuroxime axetil—β-lactamase-producing strains of <i>H. influenzae</i> and <i>M. catarrhalis</i> Erythromycin or doxycycline or cotrimoxazole—in patients sensitive to penicillin Sparfloxacin (single daily dose) b. Nasal decongestants <ul style="list-style-type: none"> 1% ephedrine or 0.1% xylo- or oxymetazoline to decongest sinus ostium and encourage drainage c. Steam inhalation (15–20 min after decongestion) <ul style="list-style-type: none"> Steam alone or medicated provides symptomatic relief and encourages sinus drainage d. Analgesics for relief of pain and headache e. Hot fomentation is often soothing and helps in resolution of inflammation 	
Operative	<ul style="list-style-type: none"> a. Antral lavage <ul style="list-style-type: none"> Done under antibiotic cover when conservative treatment fails 	<ul style="list-style-type: none"> a. Trephination of frontal sinus <ul style="list-style-type: none"> Indications <ul style="list-style-type: none"> Failed conservative therapy Increasing lid swelling Impending orbital cellulitis Procedure <ul style="list-style-type: none"> Floor of frontal sinus is exposed through 2 cm long horizontal incision in superomedial aspect of orbit below eyebrow and a hole drilled with a burr Pus is drained followed by insertion and fixation of a plastic tube for irrigation of sinus with normal saline 2–3 times daily until frontonasal duct becomes patent Patency can be demonstrated by appearance of methylene blue in nose when added to irrigating fluid
	 <p>Figure 7: Antral lavage</p>	
Complications	<ul style="list-style-type: none"> Subacute or chronic sinusitis Frontal sinusitis due to obstruction of drainage pathway Osteitis or osteomyelitis of maxilla Orbital cellulitis or abscess by spread of infection either directly from roof of maxillary sinus or indirectly, after involvement of ethmoid sinuses 	<ul style="list-style-type: none"> Chronic frontal sinusitis Orbital cellulitis Osteomyelitis of frontal bone and fistula formation Meningitis, extradural abscess or frontal lobe abscess (if infection breaks through posterior wall)

■ SHORT ANSWERS

13. Malingering.

- Malingering is deafness due to conscious effort on part of subject to deceive
- Also called nonorganic hearing loss, simulated deafness.

Etiology	Clinical features	
	Symptoms	Signs
♦ Malicious intent to claim compensation after some accident or surgery or treatment	♦ Hearing loss (usually unilateral) <ul style="list-style-type: none"> – May be total hearing loss in both ears – May be total hearing loss in only one ear – May be exaggerated loss in one or both ears 	♦ Absence of any ear pathology <ul style="list-style-type: none"> ♦ Obvious efforts to demonstrate hearing loss <ul style="list-style-type: none"> – Frequent requests to repeat question – Placing a cupped hand to an ear

Investigations (Tests to Detect Malingering)

	Procedure	Inference
a. Stenger's test <ul style="list-style-type: none"> – Very useful and reliable test Principle <ul style="list-style-type: none"> – When 2 sounds are presented simultaneously to both ears, there is an impression of sound only in ear that is nearer to sound – If an ear is deaf from any organic cause, sound will be heard in normal ear only 	♦ With eyes blindfolded (to prevent malingerer to know which tuning fork is used), present sound to ears by holding tuning forks 10 inches away from each ear <ul style="list-style-type: none"> ♦ Ask subject if he can hear and in which ear ♦ Advance fork on deaf side to 3 inches from ear and on other side to 6 inches from ear 	♦ With moving of tuning fork near to deaf side, subject will deny hearing tuning fork with normal ear
b. Chimani Moose test <ul style="list-style-type: none"> – Also called Weber's test or lateralization test – Not a reliable test Principle <ul style="list-style-type: none"> – On presentation of vibration to vertex, vibrations are localized to deaf ear in unilateral deafness but in case malingering, vibrations will be appreciated by sound ear and which would stop on occlusion of sound ear 	♦ Place a tuning fork (256Hz) on vertex <ul style="list-style-type: none"> ♦ Ask subject if he can hear and in which ear ♦ Occlude meatus of normal ear ♦ Ask subject if he can continue to hear 	♦ Subject will deny hearing in both ears
c. Lombard's test <ul style="list-style-type: none"> – Reliable test 	♦ Present a barany noise box in sound ear and accustomize subject to noise <ul style="list-style-type: none"> ♦ Ask him to read aloud from a book in his natural voice and not to stop when noise starts 	♦ In true deafness, voice of subject is markedly raised often to shout <ul style="list-style-type: none"> ♦ In malingering, subject will continue to read in same tone or only slightly raises his voice
d. Loud voice test <ul style="list-style-type: none"> – Also called Erhard's test 	♦ Present loud sound to subject from long distance <ul style="list-style-type: none"> ♦ Then occlude sound ear with a finger in meatus ♦ Present same loud sound to subject from a short distance 	♦ Subject will deny hearing test sound with meatus slightly closed even when they were heard from long distance with meatus open
e. Cochleopalpebral test <ul style="list-style-type: none"> – Also called Gault test – Useful in bilateral simulated deafness 	♦ Tightly occlude sound ear and make a loud noise near deaf ear <ul style="list-style-type: none"> ♦ Observe for winking movements or contraction of lid of corresponding eye ♦ Also observe for pupillary changes 	♦ Winking or contraction of lid on same side suggest sound was heard in that ear <ul style="list-style-type: none"> ♦ Pupils also exhibit contraction if sound is heard (auditory pupillary reaction)

	Procedure	Inference
f. Stethoscope test	<ul style="list-style-type: none"> ♦ Occlude one ear piece of stethoscope with wax and replace chest piece with funnel ♦ Present voice into funnel of stethoscope 	♦ Malingering exhibit confusion regarding voice being conducted to both ears or to single ear
g. Two speaking tubes – Useful in unilateral simulated deafness	<ul style="list-style-type: none"> ♦ Present different voices to both ears of subject by 2 different speakers with 2 different tubes ♦ Ask him to repeat what he hears 	♦ Malingering would become confused and only occasionally repeat words from one or both speakers
h. Audiometry (diagnostic)		<ul style="list-style-type: none"> ♦ Inconsistent results on repeat testing with variation greater than 15 dB (normal \pm 5dB) on pure tone audiometry and speech audiometry ♦ Stepdial reflex test better than pure tone average by >10 dB ♦ Electrical response audiometry detects simulated deafness ♦ Absence of shadow curve on testing bone conduction

14. Hematoma auris (Management).

- Hematoma auris is collection of blood between auricular cartilage and its perichondrium

Etiology	Clinical features	
	Symptoms	Signs
♦ Blunt trauma (as in boxers, wrestlers, rugby players)	<ul style="list-style-type: none"> ♦ History of blunt trauma to ear ♦ Severe pain 	♦ Tense, fluctuant swelling under auricular skin

Treatment

Conservative

- Antibiotic
 - Prophylactically
- Pressure bandage
 - For smaller hematoma

Operative

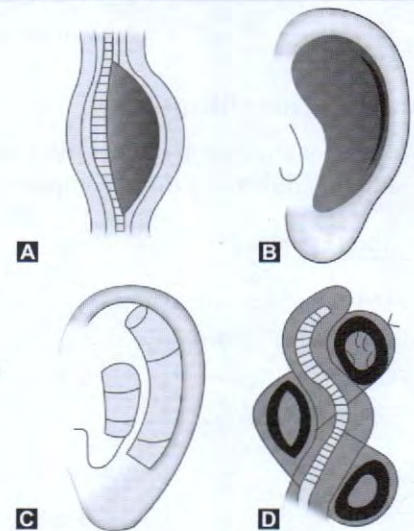
- Aspiration
 - Done under strict aseptic precautions and pressure dressing for 48 hours
- Incision and drainage
 - If aspiration fails

Complications

- Perichondritis (due to infection of hematoma)
- Cauliflower ear or boxer's ear (due organization of hematoma).

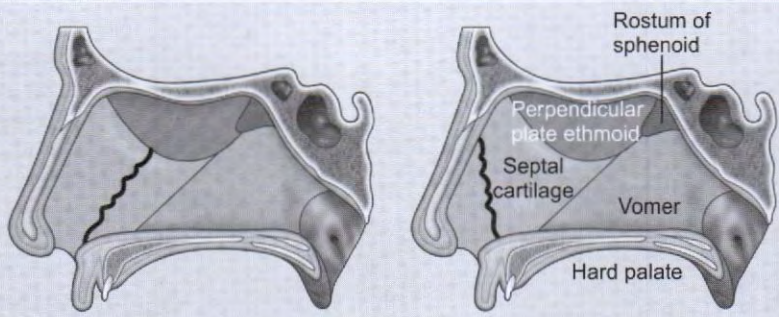
15. Nasal Septum fracture.

- Nasal septum may fracture vertically, horizontally or be crushed to pieces due to trauma inflicted on nose from front, side or below
- Septum may buckle on itself or fracture with fractured pieces overlapping each other.



Figures 8A to D: (A) Hematoma; (B) Incision; (C) Anterior dental rolls secured to posterior dental rolls; (D) Side view shows rolls are sutured

Types

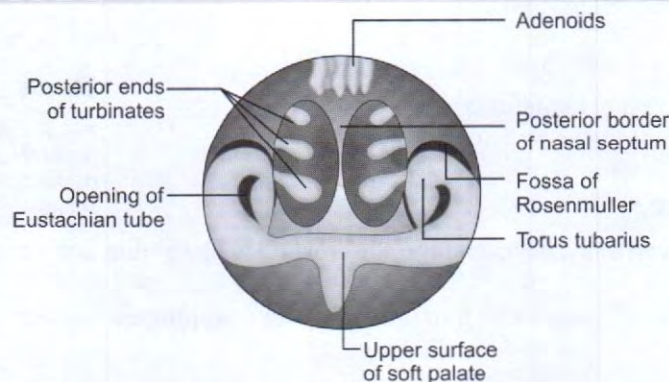
	<i>Jarjaway fracture</i>	<i>Chevallet fracture</i>
Etiology	♦ Trauma due to blows from front	♦ Trauma due to blows from below
Feature	♦ Horizontal fracture starting just above anterior nasal spine and running backwards just above junction of septal cartilage with the vomer	♦ Vertical fracture running from anterior nasal spine upwards to junction of bony and cartilaginous dorsum of nose
		
Figure 9: Nasal septum		
Clinical features	♦ Septal hematoma if mucosa is intact ♦ Profuse epistaxis if mucosa tears	
Treatment	♦ Drain the hematoma ♦ Reposition dislocated or fractured septal fragments and support them between mucoperichondrial flaps with mattress sutures and nasal packing	
Complications	♦ Deviated nasal septum ♦ Septal hematoma ♦ Saddle nose deformity due to absorption of nasal septum ♦ Asymmetry of nasal tip, columella or nostril	

16. Posterior rhinoscopy.

- Posterior rhinoscopy is a clinical procedure to directly visualize the nasopharynx using a mirror which is tilted in different directions to get complete picture.

Structures Examined

♦ Anterior wall of nasopharynx	Posterior border of nasal septum, choanae, posterior ends of turbinates and their meatuses
♦ Lateral walls of nasopharynx	Torus tubarius, opening of Eustachian tube, pharyngeal recess
♦ Floor of nasopharynx	Upper surface of soft palate
♦ Roof of posterior wall	

**Figure 10: Structures seen on posterior rhinoscopy**

Technique

- Patient sits with open mouth, facing the examiner who depresses the tongue and introduces posterior rhinoscopic mirror, which has been warmed and tested on the back of hand
- Mirror is held like a pen and carried behind the soft palate; without touching it on posterior third of tongue to avoid gag reflex, light from head mirror focussed on rhinoscopic mirror to further illuminate it.

Abnormal Findings

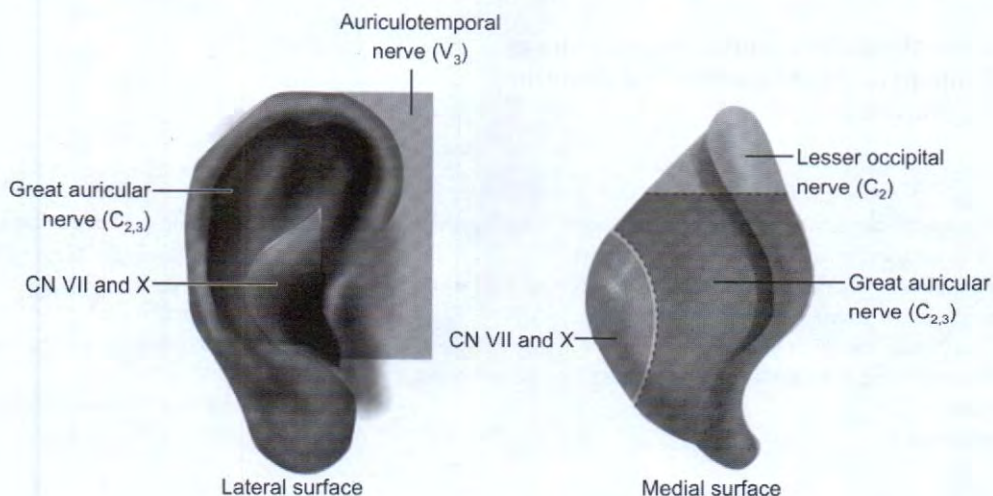
- Discharge in middle meatus
 - Seen below middle turbinate in infections of maxillary, frontal or ethmoidal sinuses
 - Seen above the middle turbinate indicates infection of posterior ethmoid or sphenoid sinuses
- Choanal atresia
- Crusting—atrophic rhinitis or nasopharyngitis
- Pathological mass
 - Smooth pale mass—antrochoanal polyp
 - Pink lobulated mass—angiofibroma
 - Irregular bleeding mass—carcinoma
 - Smooth swelling in the roof—Thornwaldt's cyst or abscess
 - Irregular mass with radiating folds—adenoids.
 - Irregular mass filling the lower part of choana—Mulberry hypertrophy of inferior turbinate
- Bleeding—due to posterior nasal or nasopharyngeal pathology.

17. Pinna—nerve supply.

- Pinna or auricle is the outer component of external ear made up of yellow elastic cartilage covered with skin (except its lobule).

Nerve Supply

Nerves	Area supplied
♦ Greater auricular nerve (C _{2,3})	Medial surface and posterior part of lateral surface
♦ Lesser occipital (C ₂)	Upper part of medial surface
♦ Auriculotemporal (V ₃)	Tragus, crus of helix and adjacent part of helix
♦ Auricular branch of vagus or Arnold's nerve (CN X)	Concha and corresponding eminence on medial surface
♦ Facial nerve	Concha and retroauricular groove (along with fibers of vagus nerve)

**Figure 11:** Nerve supply of pinna

18. Cryosurgery in otolaryngology.

- Cryosurgery is application of technique of rapid freezing of tissues to temperatures of -30°C and below and their slow thawing to cause their destruction.

Methods	Mechanism of action (tissue destruction)	Technique
a. Open method: Liquid nitrogen spray or carbon dioxide snow b. Closed system: Cryoprobe (based on Joule-Thomson effect, i.e. rapid expansion of compressed gas through a small hole produces cooling) available in different sizes and produce a tip temperature of -70°C or below. Some probes also have thermocouples which can be inserted into tissue to monitor the temperature	a. Dehydration causes cell death as follows <ul style="list-style-type: none"> – Crystallization of intracellular and extracellular pure water \rightarrow rise in electrolyte concentration – Change in pH due to crystallization of buffering substances – Raised levels of urea and dissolved gases b. Denaturation of cell membranes makes them permeable to cations which cause cell lysis c. Thermal shock arrests cellular respiratory function d. Microthrombosis of capillaries occludes arterial and venous blood supply leads to ischemic infarct e. Cryoimmunization (production of autoantibodies specific to the tissues frozen provide tissue specific immunity to prevent recurrence)	<ul style="list-style-type: none"> • Done under local or light general anesthesia or sometimes no anesthesia (as freezing itself causes numbness) • After insulation of target area including a margin of normal tissue, suitable cryoprobe is applied into or upon the tissues • Probe is frozen quickly for 3–8 min and then allowed to thaw slowly and this procedure is repeated once or twice and adequate depth of freezing is ensured using a thermocouple • Healing occurs by secondary intention with necrotic slough falling off in 3–6 weeks • Cryotherapy cycles may be repeated to achieve the desired result

Application in ENT

- Benign vascular tumors
 - Useful to treat haemangiomas involving skin, oral cavity or oropharynx
 - Also been used as an adjunct to treat vascular tumors such as angiofibroma and glomus tumor.
- Premalignant lesions
 - Cryotherapy is preferred over electrosurgery in leukoplakia, involving cheek, tongue, floor of mouth as it causes less scarring, better quality of regenerated epithelium and no recurrence.
- Malignant lesions
 - Cryotherapy is particularly useful when tumor overlies cartilage as latter does not undergo necrosis with freezing
 - Curative cryotherapy can be used in early lesions (T1 N0) involving floor of mouth, tongue and palate but with limited success
 - Major role is in palliation of advanced cancers or recurrent or residual tumors by debulking tumor mass to facilitate deglutition or respiration, reduce bleeding tendency of tumors and to relieve pain
- Other uses
 - To reduce size of nasal turbinates to improve airway
 - In allergic rhinitis to control sneezing and rhinorrhea
 - Cryodestruction of tonsils.

Advantages	Disadvantages
<ul style="list-style-type: none"> • Useful in poor risk patients, patients with bleeding disorders or coagulopathies • Can be applied without anesthesia or under local anesthesia • Can be used in multiple cancers, palliation of recurrent cancers where second course of radiation is not advisable • Causes minimal post-treatment discomfort or pain • Causes minimal scarring thus can be used at sites, notorious for keloid formation, e.g. presternal region • An outpatient procedure 	<ul style="list-style-type: none"> • No tissue for biopsy in case of small lesions • Not possible to assess margins of tumor to know whether free of malignant cells • No control on depth of freezing • Causes depigmentation and loss of hair due to destruction of hair follicles • Anesthesia required when lesion is near nerves, e.g. ulnar or digital

19. Outline differences of acute mastoiditis with Furuncle of external auditory canal (EAC).

Point of difference	Acute mastoiditis	Furuncle of external auditory canal (localized acute otitis externa)
<ul style="list-style-type: none"> History Discharge Deafness Tenderness Movement of pinna Displacement of pinna Tympanic membrane Swelling of external canal Preaural and postaural lymphadenitis X-ray mastoid 	<ul style="list-style-type: none"> Middle ear infection Mucoid to mucopurulent, profuse and pulsatile Moderate to moderately severe On mastoid antrum Not painful Downwards, outwards and forwards Congested or perforated Bony part in posterosuperior quadrant Absent Cloudiness or destruction of mastoid air cells and sclerosis 	<ul style="list-style-type: none"> Trauma, scratching Purulent (never mucoid), moderate Mild On tragus of pinna Painful Forwards Normal Cartilaginous part May be present Normal

20. Physiology of equilibrium.

- Body is said to be in equilibrium when vector sum of all torques acting on body equals zero.

Components (Balance Systems)

Sensory component	Motor component
<ul style="list-style-type: none"> Vestibular system records changes in head position, linear or angular acceleration and deceleration and gravitational effects which is sent to CNS CNS also receives information from other sensory systems, i.e. visual, auditory and somatosensory (muscles, joints, tendons, skin) CNS integrates all this information for regulation of equilibrium and body posture Cerebellum, which is connected to vestibular receptors helps in coordinating muscular movements, which vary in their rate, range, force and duration 	<ul style="list-style-type: none"> Equilibrium during standing and walking also needs motor commands, which are fine-tuned through frontal cerebral lobes, cerebellum and basal ganglia

Physiology of Equilibrium—Push and Pull System

- Physiology of equilibrium can be compared with a two-sided push and pull system
- In a neutral position, push and pull of one side is equal to that of the other side
- Balance of body is disturbed if one side is pulling more than other
- During turning or tilting, a temporary change in push and pull force of one system is taken care of by appropriate reflexes and motor outputs to eyes (vestibuloocular reflex), neck (vestibulocervical reflex) and trunk and limbs (vestibulospinal reflex), which maintains new position of head and body
- If any component of push and pull system of one side is diseased than it results in vertigo and ataxia.

21. Give differential diagnosis of lymph node in neck.

- An enlarged lymph node is most common neck swelling and need a precise differential diagnosis.

Differential Diagnosis of Enlarged Lymph Node in Neck

Inflammatory	Neoplastic	Systemic conditions
<ul style="list-style-type: none"> i. Acute nonspecific lymphadenitis <ul style="list-style-type: none"> – Viral or bacterial ii. Acute specific lymphadenitis <ul style="list-style-type: none"> – Infectious mononucleosis iii. Chronic nonspecific lymphadenitis <ul style="list-style-type: none"> – Adenotonsillar or orodental sepsis iv. Chronic specific lymphadenitis <ul style="list-style-type: none"> – Tuberculous lymphadenitis – Syphilis, leprosy, actinomycosis 	<ul style="list-style-type: none"> i. Primary <ul style="list-style-type: none"> – Lymphoma – Chronic lymphatic leukemia ii. Secondary <ul style="list-style-type: none"> – Metastasis from head and neck malignancy, GIT cancers 	<ul style="list-style-type: none"> Leukemia Niemann-Pick disease Drug reactions Epstein-Barr virus Sarcoidosis

Investigations

Panendoscopy	Radiography	FNAC and biopsy
♦ Comprises of nasopharyngoscopy, laryngoscopy, bronchoscopy and esophagoscopy to locate primary	♦ X-ray and CT scan to locate site and extent of primary	♦ Confirms diagnosis

Treatment

- Treatment of underlying cause
- Radical neck dissection.

22. Occult primary.

- Occult primary are **histologically proven** metastatic **malignant tumors** whose primary site cannot be identified during pretreatment evaluation
- Also called **cancers of unknown primary site**
- Account for 5–10% of all diagnosed cancers
- Characterized by clinical absence of primary tumors, early dissemination, aggressiveness, and unpredictability of metastatic pattern.

Etiology

- Abnormalities in p53 gene (70%)
- Chromosomal abnormalities in short arm of chromosome 1.

Clinical Features

- Common in 6th decade of life
- Equally common in men and women

Symptoms	Signs
♦ <u>Painless neck mass</u> ♦ Anorexia ♦ <u>Weight loss</u>	♦ <u>Multiple sites</u> of involvement such as lymph nodes, lung, bone, liver, pleura and brain (50%)

Treatment

Conservative	Operative
a. Radiotherapy Indications – For areas of symptomatic metastases – Lymph node metastases to cervical nodes or inguinal nodes	a. Radical neck dissection – Removal of tissues in one or both sides of neck between mandible and clavicle including all lymph nodes, jugular vein and muscles and nerves b. Modified radical neck dissection – Removal of all <u>lymph nodes in one or both sides of neck</u> without removing neck muscles but nerves and/or jugular vein may be removed c. Partial neck dissection (selective neck dissection) – Removal of some of lymph nodes in neck

Prognosis

- Median survival of about 6–9 months
- Poor prognosis in males, adenocarcinoma, hepatic involvement and an increasing number of involved organ sites.

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